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THE UNIVERSITY OF ALBERTA

THE EFFECT OF ETHANOL INGESTION ON CARDIOVASCULAR RESPONSES TO BLOWING COLD AIR ON THE FACE

BY

J. BRUCE BAIN

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF Master of Science

DEPARTMENT OF Physical Education and Sports Studies

EDMONTON, ALBERTA

SPRING, 1987

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submitted by J. Bruce Bain

in partial fulfillment of the requirements for the degree of Master of Science.

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(Supervisor)

W Haddon

A. Belanger

Date: March 31, 1987

Dedication

This thesis is dedicated to my parents, Jim and Laura Bain without whose support, love and faith this and all that has gone before would not have been possible. I would also like to dedicate this to the memory of Boris Monsaroff, my grandfather, in whose footsteps I find myself following.

Abstract

Several rapid cardiovascular responses to blowing wind at five different temperatures on a discrete area of the face and the effect of alcohol ingestion on these responses were studied. The temperatures used were 33C, 30C, 21C, 15C and 9C. Subjects were male volunteers, aged 21-32 years. The subjects were tested on two different occasions, once after ingesting alcohol (1.5 ml/kg body wt.) and once without having ingested alcohol. The order of the tests (alcohol vs no alcohol) was randomized as was the order in which the several temperatures were presented. The physiological variables measured were heart rate, arterial pressure, finger blood flow and face temperature. Mean arterial pressure and mean finger vascular resistance were calculated from the data. A thermal comfort questionnaire was also administered to assess the subjects perception of the thermal stimulus.

The results indicate that blowing air at the above temperatures on the area of the face innervated by the ophthalmic branch of the trigeminal nerve had very little effect on the variables measured with the exception of cheek temperature. In spite of a drop in cheek temperature of 10.5C when a 9C wind was used no changes were observed in heart rate or arterial pressure. Finger blood flow and mean finger vascular resistance showed a tendency to decrease and increase respectively when 9C and

15C winds were used.

Alcohol had no apparent effect on most of the variables measured although finger blood flow tended to be higher and mean finger vascular resistance lower when alcohol had been ingested. The response of mean finger vascular resistance to the wind stimuli also appeared to have been attenuated when alcohol had been ingested. Subjects also reported they felt warmer after they had ingested alcohol when air at any of the temperatures used was blown on them.

Previous studies have suggested that blowing cold wind on the face causes a rapid decrease in heart rate and an increase in peripheral resistance. It has also been suggested that this response is mediated via the trigeminal nerve and that the ophthalmic branch of this nerve is the most important. This response has been associated with the diving reflex. The results of the present study suggest that stimulation of this branch alone may not be sufficient to produce a response when are at the above temperatures is used. Additionally, in light of the modest increase in vascular resistance seen in the finger and the lack of heart rate response, it is postulated that changes observed may be as much a part of the thermoregulatory response as they are associated with a reflex similar to the diving reflex.

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I. INTRODUCTION

A. General Overview

Bradycardia in response to immersion of the face in cold water has been observed in a variety of animals including man. This has been found by some workers to be paralleled by an increase in peripheral resistance. These phenomena are often referred to collectively as the diving reflex presumably because they were first described in diving animals. In man, a similar response can be elicited by blowing cold air on the face. The responses, which occur rapidly following stimulation, apparently reflect relatively simple reflex mechanisms. However, there is evidence that these mechanisms may be affected by activity of "higher centres" of the brain, activity associated with conscious sensation and presumably perception of the intensity of the stimulus. Ingestion of ethanol has been found to reduce the perceived intensity of cold stimuli in human subjects. Whether or not this means of altering the perceived intensity of the stimulus would alter the cardiovascular responses to cold is uncertain

From studies of the response in man, Elsner et al (1963) reported both a decrease in heart rate as well as a fall in calf blood flow as subjects immersed their faces in cold water. Irving (1963) observed slowing of heart

rate in subjects when immersed in water in a swimming pool. It was still evident when they swam vigorously underwater.

The magnitude of the response has been found to be temperature dependent in man. For example Craig (1963) found in human subjects that bradycardia was more pronounced when the water was at 22C than when it was warmer (29.5-34.5C). Similarly Magel et al (1982) found a greater decrease in heart rate upon face immersion in cold (5C) than in warmer (24C) water. Wolf et al (1965) demonstrated a temperature effect in subjects using 20-40C, a range of temperatures which may not have provided a sufficiently cold stimulus to elicit the response.

The bradycardia in response to cold air on the face has been described by several authors. For example, LeBlanc et al (1975b) when studying outdoor workers (mailmen) observed a decrease in heart rate when cold air (0C) was blown on them at a rate of 40 mph. Hayward et al (1976) also reported having found a decrease in heart rate in response to cold air being blown on the face. This they found was accompanied by an increase in forearm vascular resistance as calculated by dividing mean arterial pressure (MAP) by mean forearm blood flow. Riggs et al (1981) noted a decrease in submaximal exercise heart rate at a given workload when cold air was blown on a

subject's face indicating that the response can be elicited even when the heart rate is elevated during exercise.

The bradycardia and associated responses appear to be mediated via a multineuronal reflex acting through centers in the medulla oblongata with the efferent outflows in both parasympathetic and sympathetic nerves. According to LeBlanc (1975b) the parasympathetic efferents responsible for the cardiac slowing lie within the vagus nerve. In keeping with LeBlanc's conclusion Lillo (1979) found that atropine, the action of which would block the function of the vagal endings at the heart, abolished bradycardia in bullfrogs when they were immersed in water. He also found that beta-adrenergic blockade which would inhibit the action of adrenaline at receptor sites on the heart, had no effect on the bradycardia produced by immersion suggesting little if any participation of the sympathetic nervous system in this response. Finley et al (1979) in human subjects found that atropine also reduced or eliminated the bradycardic response to facial immersion indicating the same mechanism functions in man. They concluded also that there was an increase in sympathetic tone not directly responsible for the bradycardia but for the increase in blood pressure seen during such stimulation. Such an increase would account for the peripheral vasoconstriction seen during the response by Hayward et al (1976).

The afferent fibers involved in this reflex seems to be primarily in the trigeminal nerve, specifically in the ophthalmic branch, although there may also be some input by way of the glossopharyngeal nerve. This was suggested by Andersen (1963a) after his finding that cutting the trigeminal nerve in ducks abolished the bradycardic response to immersion. He also found that, prior to nerve section, the greatest response occurred when the level of the water reached the nostrils of the ducks, an area innervated by the opthalmic branch of the trigeminal nerve. That the afferent pathway is similar in mammals was indicated by Dykes (1974) who reported that desensitization of the face of seals, either by section of the nerve or by local anaesthesia, reduced or eliminated the bradycardic response to immersion. Both LeBlanc et al (1975b) and Parfrey and Sheehan (1975) postulated that in humans the afferent pathway for this response is via the trigeminal nerve.

The pathways involved in the reflex response apparently synapse in the lower brain stem with the effect that the cerebral cortex is not essential for the response. This was indicated by findings such as those of Andersen (1963a) who found that the bradycardia could be elicited in decerebrate ducks as long as the trigeminal nerve and lower brainstem remained intact. Martner et al (1977) demonstrated a similar response in decerebrate cats. From findings such as those above one can conclude

that the response involves a basic reflex arc with afferent fibers in the trigeminal nerve and efferent fibers in the vagus nerve affecting the heart and sympathetic fibres affecting the blood vessels.

Although not essential for eliciting the bradycardic reflex the higher centers of the brain appear to exert a strong influence over its occurrence. For example, Scholander (cited by Blix and Folkow, 1977), observed that the seals on which he was experimenting would at times exhibit bradycardia even before immersion and sometimes simply upon a gesture by one of the investigators. On the other hand, reducing this influence by distracting the attention of the individual from the stimulus has been found to raise the threshold for the response. This is evident in the findings by Ross and Steptoe (1980) that having human subjects perform mental arithmetic during facial immersion reduced or abolished the bradycardic response. A more passive type of mental involvement, that of listening to reading of prose, did not seem to alter the response. Wolf et al (1965) observed that oral distraction i.e. talking or yelling, by the investigator could reduce or eliminate the bradycardia in humans during facial immersion. The same authors in a later paper (Wolf et al, 1978) noted that preoccupation with other matters attenuated or abolished the response whereas fear appeared to accentuate it. Similar observations by Furedy (1985) also indicated that physical control over the immersion

attenuated the bradycardia while knowledge of impending immersion had no effect. These types of observation suggest that mental activity, which would include that involved in one's perception of the strength of a stimulus, may act to raise or lower the threshold of the reflex response.

B. Statement of the Problem

A series of rapid cardiovascular responses to blowing cold air on the face similar to that seen during the diving reflex have been reported in humans. These responses include bradycardia, increased peripheral resistance, decreased peripheral blood flow and maintenance of arterial pressure. The number of investigations of this phenomena have been limited. The possibility that alcohol ingestion may modify these cardiovascular responses has not been investigated. Alcohol may alter the physiological responses directly or it may act by altering the subjects perception of the thermal stimulus. It has been suggested that subject response (e.g. bradycardia) to stimulation of the face by cold water is influenced by higher brain centres (Ross and Steptoe 1980, Wolf 1978).

The purpose of this study was to examine the cardiovascular responses to blowing air at a series of temperatures on the face of human male subjects and to

determine if alcohol had an effect on the nature of these responses. The physiological variables measured included heart rate, arterial pressure, face temperature and finger blood flow. Blood flow to the finger was chosen to be measured because of its easy accessibility, extreme sensitivity to environmental change and its apparent sensitivity to the subjects mental state i.e. perceived thermal comfort.

It is postulated that blowing air at the area of the face innervated by the ophthalmic branch of the trigeminal nerve, will cause a series of rapid, reflex-type cardiovascular responses to occur in. These responses may be attenuated by ingestion of alcohol which may exert a direct physiological effect or alter the subjects perception of the intensity to thermal stimulus.

II. REVIEW OF LITERATURE

A. Studies on Animals

Bradycardia induced by water immersion has been recognized in animals for many years. According to Blix & Folkow (1977) in their review on cardiovascular adjustments to diving in mammals and birds, the first observations of this phenomenon were reported in 1870 by Paul Bert, a French physiologist. Bert noticed that ducks exhibited a profound decrease in heart rate when diving. He speculated that this response combined with much larger blood volumes were responsible for the ducks much longer diving times compared to hens. Subsequently, a great deal of research has been concerned with the further characterization of the responses to diving on facial immersion in animals, as pointed out by Blix and Folkow (1977). In general, these responses have been found to include the bradycardia mentioned above accompanied by an increase in peripheral resistance and an associated decrease in peripheral blood flow. Blood pressure is generally maintained or slightly elevated even during periods of extremely low heart rates as indicated in a review by Scholander (1962).

The aforementioned responses to diving appear to be the result of an underlying reflex arc with afferent fibres originating in the ophthalmic and maxillary

branches of the trigeminal nerve. This conclusion is based on findings such as those by Andersen (1963) in ducks and has been corroborated by others including Dykes (1974) who performed experiments on seals. The complete pathway is reviewed below (see p. 20).

The decrease in peripheral circulation in seals has been graphically demonstrated by Scholander as cited by Blix and Folkow (1977). A seal was subjected to head immersion and a section of skeletal muscle exposed. When this piece of muscle was cut, no bleeding occurred. When the dive was terminated the cut muscle bled profusely. In keeping with this, Grogaard & Sundell (1983) found that holding a cold wet towel against the snout of newborn lambs decreased aortic blood flow by an average of almost 25% below control levels. Interestingly, carotid flow increased although only by an average of about 8% suggesting that flow to the head was maintained.

A decrease in heart rate is the most commonly observed phenomenon in response to facial immersion. Scholander, in his 1962 review, noted that this response has been shown to occur in mammals, birds, reptiles, amphibians and even fish when removed from water. As a specific example, Lin (1974) observed that slowing of the heart rate occurred in rats when their heads were immersed in water. Both Andersen (1963) and Langille (1983) have made similar observations using ducks.

In addition to decreases in heart rate there is apparently a decrease in cardiac output during diving as well. Blix and Folkow (1977) cite several references supporting this. In keeping with these studies, a more recent investigation (Grogaard & Sundell, 1983) has shown a decrease in cardiac output of approximately 20% in newborn lambs on application of cold to the snout. In his 1982 review, Lin states that there is apparently no significant change in stroke volume. According to Blix and Folkow, the decrease in heart rate and cardiac output along with the maintenance of stroke volume indicates a reduction in myocardial contractility. It should be noted here that the circumstances surrounding the experiment may also affect the responses seen. For example, Butler in a review published in 1982 indicates that free diving experiments have shown a much greater variability in the heart rate responses in both mammals and birds. This may indicate influences of other factors such as fear in captive experiments, which infers possible participation of higher brain centres as modifiers of a basic reflex.

As indicated above, arterial pressure remains the same or is slightly elevated during diving or facial immersion. For example, an increase in both systolic and diastolic pressure was found in newborn lambs during simulated diving (Grogaard and Sundell, 1983). Lin (1974) found a similar response in rats. Similarly Lillo (1979) found that arterial pressure was maintained or

slightly elevated in bullfrogs when they were submerged in water. This response was also observed in decerebrate cats (Martner et al, 1977).

In summary, it would appear that there is a general response to diving or facial immersion seen in most animals which is at least partly characterized by a decrease in heart rate with associated changes in cardiac performance, an increase or maintenance of arterial pressure, and a decrease in peripheral blood flow. These responses appear to vary in intensity between species and also depend on the circumstances of the particular experiment.

B. Studies on Human Subjects

Face Immersion in Water

In line with the research carried out on animal models concerning the various cardiovascular responses to diving and/or facial immersion, studies have been carried out by numerous investigators using human subjects to determine if the same reflex is present in man and to elucidate the underlying mechanisms by which they are triggered. Among the earlier investigations of this response were again carried out by Scholander et al (1962). For example, they found a decrease in heart rate in free diving pearl divers during underwater swimming. Arterial pressure measured during facial immersion while

at the surface showed little change.

To obtain an indication of muscle blood flow during the dive, Scholander and co-workers (1962) measured pre and post dive blood lactate values. They found that lactate values in blood taken immediately after diving while subjects were still holding their breath were the same as pre-dive values. Blood samples taken following the resumption of respiration had much higher lactate levels, a response similar to that found in seals. They concluded that this delayed increase in lactate possibly indicated a decrease in muscle blood flow during a dive which resulted in a buildup of lactate in the muscle. The increase seen in lactate values after breathing was resumed was interpreted as reflecting an increased muscle blood flow which essentially washed the lactate out of the muscle into the circulation.

The bradycardia response has also been found to be accompanied by a reduction of blood flow to various parts of the body as has been suggested above (Scholander, 1962). For example, Elsner (1963) found a decrease both in heart rate and calf blood flow during facial immersion. In keeping with this, Heistad et al (1968) found a decrease in heart rate and finger and forearm blood flow as well as an increase in arterial pressure in human subjects during facial immersion. The responses in fact were quite marked. For example, finger blood flow

decreased by 72% within the first ten seconds of immersion while forearm flow decreased by about 50%. Mean arterial pressure increased approximately 20% while heart rate decreased approximately 23%. In fact similar responses have been found in varying degrees by many authors (see Table I, pg. 14).

TABLE I CARDIOVASCULAR RESPONSE TO COLD ON THE FACE IN HUMANS

Author / Yr	Stimulus	Response			
		HR	BP	BF	VR
Kawakami et al, 1967	Breath Hold, Face Immersion	▼	▲	--	--
Furedy et al, 1983	FI water 10-40C	▼	--	▼	--
Leblanc J, 1975	FI water 30C & 4C cold air	▼ 4C H_2O + cold air	▲	--	--
Elsner et al, 1963	BH & FI	▼	--	▼	--
Irving L, 1963	I & swim submerged	▼	--	--	--
Hardy et al, 1965	BH, I to neck	--	▲	--	--
Craig A.B., 1963	BH, I	▼	--	--	--
Whayne et al, 1972	0C cloth on face	▼	▲	--	--
Furedy J.J., 1985	FI	▼	--	--	--
Moore et al, 1972	BH & FI varied water temp.	▼	--	--	--
Gandevia et al, 1977	FI	▼	--	--	--

▲ - increase
▼ - decrease
-- - same or not measured

BH - breath hold
FI - face immersion
I - immersion

HR - heart rate
BP - blood pressure
BF - blood flow
VR - vascular resistance

Author / Yr	Stimulus	Response HR	BP	BF	VR
Folgering et al, 1983	FI	▼	--	--	▲
Olsen et al, 1962	BH & total water immersion water temp varied	▼	--	--	--
Khurana et al, 1980	BH, cold compress on face, FI	▼	▲	--	--
Wolf et al, 1965	FI	▼	--	--	--
Mukhtar & Patrick, 1984	BH & FI, 10C H_2O	▼	--	--	--
Scholander et al, 1962	free dive in ocean	▼	no change	--	--
Heistad et al, 1968	FI, 20-25C H_2O	▼	▲	▼	--
Parfrey & Sheehan, 1975	BH & FI, 5-30C H_2O	▼	--	▼	--
Stromme et al, 1970	BH, FI	▼	--	--	--
Folinsbee L., 1974	BH & FI, 35C-30C H_2O	▼	▲	▼	--
Riggs et al, 1981	cold wind on face 2 & 10C	▼	--	▼	--
Speck & Bruce, 1978	BH & FI	▼	▲	--	--
Cabanac & Caputa, 1979	cold wind, 10C	▼	--	▼	--

Author / Yr	Stimulus	Response HR	BP	BF	VR
Leblanc et al, 1975b	H_2O 5C FI, wind 0C, 40 mph	↓	↑	--	--
Hayward et al, 1976	0-10C air	↓	little change	↓	↑
Ross & Steptoe, 1979	BH, FI, H_2O 15C	↓	--	--	--
Furedy J.J., 1985	BH, FI	↓	--	↓	--
Nagel et al, 1982	BH, FI H_2O 24C, 5C	↓	--	--	--
Olsen et al, 1962	Supine Immersion H_2O, 26C & 37C	↓	↑	--	--
Smeland et al, 1984	BH, FI	↓	--	--	--
Finley et al, 1979	FI	↓	↑	--	--

The magnitude of the responses seems to be at least partly dependent on both water temperature and whether or not subjects are holding their breath. Breath holding alone seems to produce at least some slowing of the heart. For example, Moore et al (1972) observed a slight decrease in heart rate when subjects held their breath in air. This has been demonstrated by many other authors (Olsen et al 1962, Elsner 1963, Irving 1963, Kawakami et al 1967, Paulev 1968). The combining of breath holding and facial immersion seems to magnify the response. This finding is apparent in the work of Khurana et al (1980) who found that the greatest decrease in heart rate was seen when breath holding and facial immersion were combined as compared to breath holding alone and facial immersion while breathing through a snorkel. This is supported by various authors including Mukhtar and Patrick (1984) and Speck and Bruce (1978). Based on such evidence it appears that bradycardia induced by diving is produced by two stimuli which will act on their own or in combination.

Arterial pressure also appears to be affected to different degrees with respect to these various stimuli. For example, Khurana et al (1980) indicate that mean arterial pressure rose significantly during breath hold in air and breath hold during face immersion. Facial immersion while breathing using a snorkel, thus avoiding breathholding, did not produce a significant increase in mean arterial pressure however, whereas application of a

cold stimulus in the form of an ice bag did.

The effect of water temperature on these responses has also been examined. In general, most authors have found that colder water produces a greater response. For example, Kawakami et al (1967) found that subjects showed a greater decrease in heart rate and increase in blood pressure in cold water than in cool water. Similarly, Furedy et al (1983) examined the effects of different water temperatures ranging from 10C to 40C. They also reported that decrease in heart rate and finger blood flow (as measured by finger pulse volume) were greater in the group exposed to water at 10C. This has been confirmed by many authors. (Table I pg. 14)

This indicates that the intensity of cold may be an important factor in producing the so-called diving response in humans. This has not been found to be the case in animals. This discrepancy prompted Paulev (1968) to question the supposed similarity of the observed responses in humans and animals. Whether or not these reported responses to breath-holding and facial immersion are the same in humans as in animals, it seems clear that some kind of reflex-like reaction takes place in response to these two stimuli alone or in combination.

Cold Air on the Face

There are many reports that similar heart rate and

blood pressure responses have been seen in man when cold air is blown on the face. For example, Paulev (1968) observed a decrease in heart rate of 4% in subjects lying prone with 21C air blowing on their faces. Similarly, Hayward et al (1976) reported a decrease of 6±1% in heart rate when cold air was blown onto the faces of subjects sitting upright on a couch. No change was observed in mean arterial pressure in this study, but forearm blood flow, measured by strain gauge plethysmography was seen to decrease while forearm vascular resistance increased by a mean value of 30%. Similar but greater responses occurred when air was blown onto the side of the face. In keeping with these results, LeBlanc et al (1976) also reported a decrease in heart rate upon application of a cold stream of air on the face. These authors reported similar findings in a paper published in 1975.

The degree of slowing of the heart in response to cold air blown on the face appears to be temperature dependent. Paulev (1968) for example, noted a decrease in heart rate of 2.4% when 35C air was blown onto subjects' faces and a decrease of 4% when colder (21C) air was used. LeBlanc et al (1976) also noted that varying air temperatures from 25C to -15C at a given wind speed produced progressively greater decreases in heart rate as air temperature decreased. Cabanac and Caputa (1979) reported a decrease in heart rate and a mild vasoconstriction (as inferred by finger temperature) in

subjects exercising on a bicycle in a cold (10C) chamber when the face was fanned. Similarly Riggs et al (1981), reported a decrease in exercise heart rates in subjects pedaling a bicycle during incremental work when cold air (10C) was directed on the face as compared to control (no air) conditions.

One can conclude from these studies that cool or cold air blowing on the face in humans can produce similar responses to those observed during face immersion and/or breathholding or other cold stimuli applied to the face i.e. ice packs. The fact that there seems to be a temperature effect indicates a role of cold receptors in the skin although Cabanac and Caputa has postulated that the response may be related to selective brain (presumably hypothalamic) cooling via blood collected from the orbital region of the eye by the angularis oculi veins.

C. Mechanisms Underlying the Response

The cardiovascular responses to facial immersion seen in various species including man, are thought to be mediated by way of a multineuronal reflex with synapses in the medulla oblongata. The primary afferent pathway is thought to be in the trigeminal nerve via nerve fibers in the opthalmic branch. The efferent pathway is believed to be by way of the vagus nerve and sympathetic fibres originating from the spinal nerves T1 to L2 (Berne and

Levy, 1972).

Evidence that the primary afferent pathway lies within the trigeminal nerve has been put forth by many authors. For example Andersen (1963a) found that immersion of ducks heads in water produced the greater bradycardia only after the nostrils were covered. This area of the ducks bill (corresponding to the nasal and eye area in humans) is innervated by the opthalmic branch of the trigeminal nerve. This was supported by Andersens' findings that cutting the trigeminal nerve abolished the response. In keeping with this, Dykes (1974) found that if the trigeminal nerve in seals was sectioned or the area of the face supplied by that nerve anesthetized, the response to facial immersion was attenuated or abolished. Evidence that the afferent pathway of the reflex is also by way of the trigeminal nerve in humans is provided by Khurana et al (1980) who found that cold compresses (i.e. cloth soaked in ice water) applied to the area of the face innervated by the opthalmic branch of the trigeminal nerve produced a greater decrease in heart rate than that produced by application to the areas innervated by either the maxillary or mandibular branches of the same nerve.

It has been noted by many authors (Brick 1966, Olsen 1962) that breath-holding produces some bradycardia without any simultaneous stimulation of the trigeminal

nerve. Therefore it would seem that inhibition of respiration alone provides a type of stimulus which causes this reduction in heart rate. In his review, Lin (1982) states that this observed reduction in heart rate is at least partially due to the elimination of the slight tachycardia that would otherwise occur during inspiration. Blix and Folkow (1977), in their review of cardiovascular adjustments to diving in mammals and birds, concluded that receptors around the glottis, innervated by the glossopharyngeal nerve seen to also play an important role in the elicitation of bradycardia. Stimulation of these receptors causes the reflex closure of the glottis and apnea with a concommitant reduction of heart rate. They also indicate that other papers report that sectioning of these nerves did not completely abolish the decrease in heart rate seen upon stimulation of the trigeminal nerve endings, again suggesting that the trigeminal nerve fibres are an important afferent pathway in the mediation of bradycardia seen during diving.

Other stimuli can also elicit a bradycardia. For example, pressure on the eyeballs has been shown to cause such a response. This phenomenon is referred to as the oculocardiac reflex by Gandevia et al (1978). These authors reported bradycardia in response to both pressure on the eyeball and facial immersion. They postulated that these two different stimuli might have the same reflex pathway. The oculocardiac reflex has apparently been used

to predict the degree of bradycardia that might be expected in scuba divers, however, Folgering et al (1983) found a low correlation between the heart rate responses elicited by the two stimuli.

The efferent pathways by which the cardiovascular responses to facial immersion are affected appears to be contained in both the parasympathetic and sympathetic nervous systems as mentioned previously. The slowing of the heart rate seems to be mediated by way of parasympathetic fibres in the vagus nerve. This has been elucidated in studies using atropine, a drug which is used to block parasympathetic postganglionic receptor sites at the heart. For example, Lin (1974) reported that administration of atropine to block the action of parasympathetic fibres in the vagus nerve leading to the heart, decreased the heart rate response to immersion in rats. Similar effects were seen in bullfrogs by Lillo (1979). Several workers including Heistad et al (1968) and Finley et al (1979) have reported similar results in humans.

Although slowing of the heart could possibly reflect reduced sympathetic stimulation the evidence seems to be to the contrary. Various workers have found other cardiovascular responses to facial immersion such as blood pressure can be changed using drugs such as propranolol and phentolamine. These drugs affect the action of the

sympathetic nervous system. Propranolol blocks the beta-receptors in various tissues such as the heart while phentolamine blocks the alpha-receptors in tissues such as the blood vessels in the skin. For example, Finley et al (1979) found that administration of propranolol did not affect the heart rate response to facial immersion in humans but prevented the usual rise in arterial pressure. Administration of phentolamine, which should prevent a rise in diastolic pressure by preventing vasoconstriction in the skin, had no effect on heart rate response to immersion, suggesting that increases in diastolic pressure are not necessary to produce bradycardia. In addition, Heistad et al (1968) found that administration of atropine abolished bradycardia during face immersion, it did not affect the vasoconstrictor response in forearm and fingers.

Evidence for an increase in both parasympathetic and sympathetic outflow to the heart and the relative predominance of parasympathetic activity has recently been put forth by Furedy (1985). This author has suggested that changes in the amplitude of the T-wave (TWA) of the ECG, reflect changes in sympathetic activity in the heart. He reported that facial immersion resulted in an attenuation of TWA in human subjects. According to the author, a reduction in TWA indicates an increase in sympathetic activity to the heart. In spite of this suggested increased sympathetic activity, heart rate still

declined during immersion. The author concluded that although sympathetic activity had increased as indicated by the attenuated TWA's, parasympathetic activity must have been overriding this, because the heart rate decreased.

Although activity of the parasympathetic nerves seem to exert control effect over heart rate during the response to cold stimulus to the face, the stimulus might increase sympathetic activity which, while not affecting heart rate, may cause other changes in cardiac performance. As indicated above, Furedy (1985) concluded that there is an increase in sympathetic discharge to the heart during facial immersion. This may be supported by evidence given by LeBlanc (1975a) who reported that exposure of the face to cold wind results in an increase in ventricular performance as indicated by an increase in left ventricular ejection time (LVET) and a decrease in pre-ejection period (PEP). This however, is not in keeping with Lin (1974) who found a decrease in left ventricular power of 61% in rats during head down immersion.

From the above discussion, the cardiovascular responses to diving, face immersion or application of cold to the face appears to be a reflex. This appears to be fairly certain in animals such as the seal and the duck, both aquatic animals as well as the cat, which is

definitely non-aquatic. For example, Andersen (1963) states that decerebrate ducks demonstrated a functioning response to facial immersion, suggesting a basic reflex pathway capable of functioning without cerebral influence. Similar results were found by Martner et al (1977) in decerebrate cats, animals which inherently avoid water. The evidence is less clear for the mechanism in humans. For example, Asmussen and Kristiansson (1968) suggest that face immersion bradycardia may be simply a conditioned reflex developed from an unconditioned apneic inhibitory reflex'. In other words, the diving response seen in animals may not really have a counterpart in humans, rather it may be an artifact of some other reflex. However, results from other studies using various methods of eliciting this so-called diving reflex, such as face immersion, cold air, cold compresses etc. would preclude dismissing the idea of a basic reflex.

Higher brain centres also appear to play an important role in the modification of the reflex ever in nonhuman species. For example, Scholander (cited by Blix and Folkow, 1977) noted that seals he was studying would sometimes exhibit bradycardia prior to immersion and occasionally even upon a simple gesture by one of the investigators. Similarly others cited by Blix and Folkow have indicated that animals allowed to engage in free diving, that is unrestrained and of their own accord, did not always exhibit the response, a discovery which sparked

some controversy which still exists today. This suggests that higher brain centres may in some cases facilitate and in others inhibit the reflex.

In humans, various types of mental activity or distraction have been found to attenuate the response. As indicated previously, Ross and Steptoe (1980) noted that performing mental arithmetic during face immersion attenuated or abolished the bradycardia seen during control periods. Listening to prose being read did not seem to affect the response. Similarly, Wolf et al (1965) noted that distracting subjects during face immersion also reduced or abolished the heart rate response. In a later paper, of 1978, the same authors found that preoccupation with other matters had a similar effect while fear accentuated the heart rate response to immersion. In a similar study, Furedy (1985) noted that if subjects were able to physically control the time of face immersion, the heart rate response was attenuated. However, simply knowing when immersion was to occur did not seem to affect the response. Thus it appears that higher centres of the brain can exert an important influence over what seems to be a basic reflex mechanism.

D. Effects of Alcohol on Various Systems of the Body

Central Nervous System

Possibly the most profound effect of alcohol is on

one's mental function, involving higher centres of the CNS, in particular the cerebral cortex, Ritchie (1975) describes alcohol as a central depressant. Although most individuals would describe the subjective effect of drinking small amounts of alcohol as being one of stimulation, Ritchie interprets its effect as being more a depression of normal inhibitory or controlling influences of the higher centres. He describes the effect as being due to depression of polysynaptic structures of the reticular activating system and certain cortical mechanisms which in effect acts to release the cerebral cortex from integrative control.

One's perception of sensory stimuli can also be altered by ingestion of alcohol. Such an effect is indicated by findings such as those of Gurney (1983). He found subjects constantly judged the temperature to which they were exposed to be warmer when they had first ingested alcohol. Similarly, Keatinge and Evans (1960) found that although ingestion of ethanol (75mL absolute) had little effect on heat loss when immersed in cold water their subjects reported that the water felt warmer after they had drank the alcohol.

Alcohol may also affect the function of peripheral nerves directly. This was indicated in the findings of Sauerland et al (1970) who examined the effects of alcohol on the stimulation threshold of the trigeminal nerve.

They found that blood alcohol levels greater than 50 mg% caused depolarization of sensory neurons in the trigeminal nerve, decreasing nerve conduction. The authors concluded that alcohol caused a presynaptic inhibition which is at least partially responsible for its central depressant effect.

Ingestion of alcohol has also been shown to act at the spinal level altering the threshold of some spinal reflexes. According to Ritchie (1975) small amounts of alcohol appear to facilitate some reactions whereas ingestion of large quantities causes elevation of their thresholds.

The effects of alcohol on neurons of the nervous system are likely the result of alteration of certain functional characteristics of the cell membrane in the neuron. For example Grenell (1972) in a review, indicated that alcohol seems to interfere with the generation of the action potential by the neuron. This is apparently caused by a change in membrane permeability to sodium and potassium which may occur as a result of changes in the levels of extracellular calcium. As neuron excitability decreases in the presence of high concentrations of extracellular calcium and, according to the author, membrane conductance decreases in the presence of ethanol, it is possible that ethanol somehow increases the level of extracellular calcium. The author concludes that synaptic

transmission is affected as a result of these and other changes. This is in agreement with the conclusions of Sauerland et al (1970) above.

Cardiovascular System

The ingestion of alcohol appears to affect the heart directly, both in terms of heart rate and contractility. The results of studies which have examined the effects of alcohol on heart rate and cardiac performance (i.e. cardiac output, stroke volume, contractility) in both humans and animals are somewhat conflicting. Although it is commonly assumed that alcohol ingestion causes an increase in heart rate, experimental evidence does not always bear it out. Regan et al (1971), in a review examining the effects of alcohol on the heart, cites only two studies that indicate that acute alcohol consumption increases heart rate in humans. Segal et al (1984) in their review of the effects of alcohol on the heart, concluded that moderate doses of alcohol have a minimal effect on the mechanical properties of the heart. According to these authors, alcohol may increase heart rate and depress myocardial contractility in human subjects. Ahmed et al (1973) found that, low doses of alcohol (6 oz whiskey ingested over a 2 hour period or a 1 hour period) produced a decrease in myocardial contractility in humans. According to the authors, this was indicated by an increase in the ratio of pre-ejection period (PEP) to left ventricular ejection time (LVET).

These measures have previously been shown to be valid indicators of cardiac performance and myocardial contractility (Ahmed et al 1972, Weissler et al 1961). Although in the same study, Ahmed et al (1973) stated that the heart rate was not elevated by a statistically significant amount, examination of the data reveals a 4% and 2% increase in heart rate over the 2 hour and 1 hour periods respectively. In keeping with this, Friedman et al (1979) found that infusion of alcohol into dogs caused a decrease in an index of myocardial contractility (as calculated by dividing the first derivative of left ventricular pulse pressure (dP/dt) by left ventricular end diastolic volume (EDV), both measured invasively and an increase of myocardial oxygen consumption. Although heart rate was not determined by the authors, examination of the data indicates that alcohol treated animals had slightly higher heart rates than animals which had not been infused with alcohol. Since heart rate has been shown to correlate well with direct measures of myocardial oxygen consumption and coronary blood flow (Hedworth-Whitty et al 1970, Kitamura et al 1972), an increase in heart rate might have occurred as they found an increase in myocardial oxygen consumption. In contrast to this, Child et al (1979) concluded that ingestion of moderate amounts of ethanol did not result in any significant effect on cardiac function in humans. However, examination of their data shows that heart rate was increased by about 7% over control values and the ratio of PEP/LVET showed a slight

increase, suggesting a small decrease in myocardial contractility. However, they also examined the effect of alcohol ingestion on cardiac performance under autonomic blockade induced by the drug propranolol, which would block the stimulatory effect of sympathetic activity, and atropine, which would block the inhibitory effect of stimulation via the parasympathetic system. Ingestion of alcohol with the autonomic blockade produced an increase in heart rate of 43% over control values. However, this represented a 10% decrease when compared to heart rate measured under autonomic blockade alone. Measurement of myocardial contractility (as calculated by the ratio PEP/LVET) under the conditions of both alcohol and autonomic blockade and blockade alone, revealed an increase in the ratio of PEP/LVET of 4% when the alcohol and autonomic blockade condition was compared to blockade alone. These authors concluded that this indicated a decrease in left ventricular performance which suggested a decrease in myocardial contractility. They speculated further that the absence of any observable significant effect of alcohol on cardiac performance in normal human subjects is likely due to compensation by the autonomic nervous system. In support of this they stated that alcohol has been known to increase the levels of circulating catecholamines which may enhance this compensation. In summary they concluded that alcohol tends to reduce the force of contraction of cardiac muscle (negative intropic effect) but that this effect is masked

by an ANS and circulating catecholamines.

On the basis of the above findings it would appear that low to moderate doses of alcohol may have some direct influence on cardiac function in normal individuals. This is not always readily apparent due to influences of other factors such as the action of the autonomic nervous system and of catecholamines circulating in the blood.

Peripheral Circulation

Peripheral circulation, in particular that of muscle and skin, may also be affected by alcohol. Ingestion of alcohol has been shown to increase blood flow to the skin in the fingers, hands, legs and forearms. For example, Gillespie (1967) reported a greater skin blood flow in the wrists and feet of twelve healthy subjects who had ingested whisky (2 mL/kg body weight). The author did not record ambient temperature. Blood flow to the muscle, measured in the calves and forearms was not increased. Blood flow in this experiment was determined by venous occlusion plethysmography using mercury in rubber strain gauges. This is in keeping with the earlier findings of Fewings et al (1966) who reported an increase in blood flow to the hand and in skin flow in the forearm but a decrease in blood flow to the muscle in the forearm. These measurements were also made using venous occlusion plethysmography but using a water-filled plethysmograph for the hand and capacitance plethysmographs (using a

copper cylinder and the skin as plates of the capacitor) for the forearm. Differences in skin and muscle flow in the forearm were determined by determining the oxygen saturation of blood taken from both skin & muscle veins via catheters. Both groups of investigators reported that intra-arterial infusion of alcohol caused a vasoconstriction. They postulated that partial metabolism of alcohol must occur for vasodilation to occur. Hughes et al (1984) also report a greater skin blood flow in the lower legs of males who had ingested alcohol. Skin blood flow, as determined by the Xenon-133 skin perfusion technique, was observed to increase after the ingestion of alcohol at environmental temperatures of 25 and 30C. This change in skin blood flow was not seen at temperatures of 20C or 35C. The authors concluded that, with sufficient cold stimulus, the vasodilatory effects of alcohol may be overridden and with sufficient heat stimulus, vasodilation is maximal and will not be affected by alcohol. The effect of cold on skin blood flow response to alcohol ingestion observed by these authors is in keeping with that seen by other investigators. For example, Andersen et al (1963) reported that ingestion of 1.1 to 1.5 g/kg of alcohol by male subjects who then rested for 8 hours in a room at 20 or 15C did not cause an appreciably greater heat loss than when alcohol was not ingested. They concluded that the cold stress at these temperatures was enough to counter any vasodilatory effects of alcohol. Similar results have been found by other workers (Fellows

et al 1984, Lomax et al 1981).

From such evidence one can conclude that ingestion of relatively moderate amounts of alcohol may cause an increase in skin blood flow in normal subjects at near to or thermoneutral ambient temperatures. This effect may be due in part to the affect of alcohol on the CNS, however Gillespie (1967) observed a greater skin blood flow in the upper limbs of recently sympathectomized patients after alcohol ingestion.

III. METHODS AND PROCEDURES

A. Subjects

Ten males 21-32 years, all students in the Department of Physical Education and Sport Studies at the University of Alberta acted as subjects. Each took part in two experimental trials. Prior to the first trial, each subject was informed of the requirements and risks involved after which each signed a statement indicating that they understood and agreed to the conditions of the experiment. (Appendix III)

Subjects were requested not to ingest any food, consume any beverages containing caffeine for at least 3 hours on the day of each trial and were not to have consumed alcohol, smoked cigarettes or taken drugs for 24 hours. They were also requested not to take part in strenuous physical activity for at least 6 hours prior to the experiment. All tests were carried out between 8:00 am and 12:00 noon.

B. Apparatus and Techniques

Air flow was supplied via a boxlike air duct through by means of a blower attached at one end. Inside, the duct was divided into two channels in one of which was mounted a heat exchanger through which cold air could be

circulated to cool the air and in the other, an electric heating coil to heat it. By moving a sliding damper the relative amount of the air passing through each channel could be controlled, making a wide range of temperatures (33C to 9C) available. The air next entered a space where mixing could occur. After passing through a screen (painted white so subjects could not see the damper) it exited via a cone-shaped duct through a square opening measuring 10 cm * 10 cm. The air velocity was approximately 8m/sec at the open outlet. The outlet had a strip of foam padding attached to the top edge against which the subjects were instructed to place their foreheads. This standardized the distance of their faces from the opening.

Heart rate and blood flow were recorded on a polygraph (Dynograph, model R, Beckman Instr. Co.). Heart rate was monitored using a cardiotachometer coupler. This latter device determines the interval between successive R-waves of the ECG and updates each beat. The cardiotachometer output, recorded on the polygraph, resulted in a series of horizontal lines on the chart each of which represented a specific heart rate. The cardiotachometer was calibrated prior to each experiment using a simulated ECG generator (Quinton Instr. Inc.).

Finger blood flow was determined by venous occlusion plethysmography using the method similar to that described

by Greenfield et al (1963). In this procedure venous drainage from the finger is occluded and the resultant change in finger volume determined. The initial rate of increase in volume is assumed to be the result of and equivalent to the rate of blood flow into the finger. Volume changes were recorded by means of an oncometer sealed over the finger slightly distal to the second finger joint using a silicon-based dental impression material (Permlastic, Kerr Inc.). The small nipple on the oncometer was attached via small bore latex rubber tubing to a low-pressure transducer (Grass Instr. Co. Model PT-5). The output of the transducer was recorded on the polygraph. A sample record may be seen in fig. 1. The system was calibrated for each subject by injecting a known volume of air into the system with the finger in place. A small inflatable cuff was placed about the base of the finger and held in place by velcro strips was inflated to a pressure of 70 mmHg during each determination to prevent venous drainage while presumably not affecting arterial flow to the finger.

The sequence of events for each blood flow determination was initiated by a push button switch and proceeded automatically. This included:

1) Increase in chart speed from 25 mm/min to 25 mm/sec.
2) Inflation of the occlusion cuff.

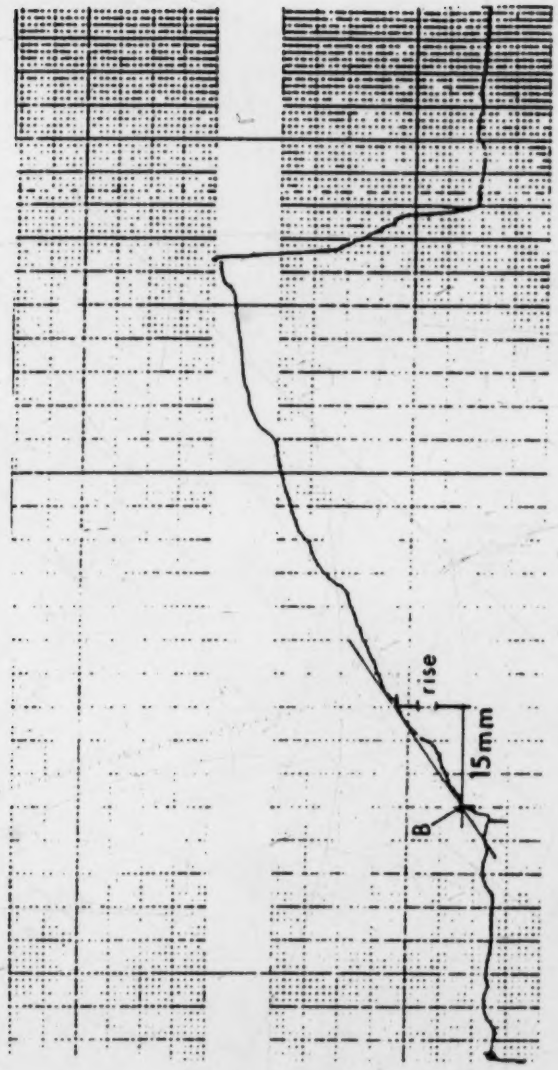

Fig.1. Sample blood flow tracing. Rate of increase in finger volume was calculated from the initial slope of each curve. Slope was calculated by drawing a tangent over the curve beginning immediately after artifact (point B) for 15 mm and counting the rise over this distance.

3) A 5 sec recording period.

4) Release of cuff pressure and slowing of chart speed to 25 mm/min.

Blood flow was measured 3 times during the first fifteen seconds of each minute of a two minute period during which wind at each specific temperature was blown on the subjects face. It was measured in the same fashion for each minute of a three minute initial no wind, or baseline, period at the beginning of each trial and during the final minute of a control period between different wind temperatures.

Arterial pressure was measured at the same time as blood flow and was determined using an automatic sphygmomanometer with as digital readout (Arteriosonde). It was only possible to measure arterial pressure once each minute as this procedure took 45-50 seconds. The cuff for this was placed on the arm (left), opposite that in which blood flow determinations were made, midway between the axilla and the elbow. Mean arterial pressure was estimated using the equation MAP = diastolic pressure + (1/3 pulse pressure).

Cheek temperature was monitored to assess the degree of cooling of the face during wind exposure. For this, a

fine thermocouple of #30 gauge wire was attached to the cheek just inferior to the left orbit by means of a small square of adhesive tape (Micropore, 3M Inc.). The thermocouple was connected to an electronic thermometer with a digital readout (Bailey Instr. Co. BAT-8).

Blood alcohol levels were estimated from breath samples using a Breathalyser (model 900A Smith & Wesson, General Ordnance Equipment Co.). Measurements were taken immediately before testing began and again at the end of the experiment.

Mean finger vascular resistance (MFVR) was calculated in order to obtain a better index of vascular tone in the fingers. As resistance is determined by the relationship between pressure and flow it may better indicate the state of the blood vessels i.e. degree of constriction in the fingers. A decrease in flow may be a result of a drop in cardiac output or vasoconstriction. Calculation of the resistance would hopefully eliminate the former. MFVR was calculated by dividing mean arterial pressure (MAP) by finger blood flow (FBF).

C. Procedures

Upon arrival in the laboratory, an oncometer appropriate to the size of the subjects right, middle finger was positioned on the finger so as to obtain

maximal coverage without blocking the nipple. A line was drawn on the subjects finger to indicate the position of the proximal end of the oncometer. The oncometer was removed and the volume of the subjects finger was then determined. For this the subject lowered his finger, to the level of the line previously drawn, into a 100 ml graduated cylinder filled with water. On removal the water displaced by the finger (finger volume) was read from the scale. Following this, subjects were given a volume (750 ml) of liquid to drink. This consisted of diet type ginger ale or a mixture of this ginger ale and vodka (Smirnoff's Blue Label 48.5% alc/vol). This was consumed over a period of half an hour. The amount of alcohol added was determined by multiplying the subjects weight in kg. (obtained previously) by the desired dose level (1.5 ml/kg body wt.). The drink was served cold. The subjects were not informed as to which mixture they would be ingesting. The order of the trials (alcohol vs no alcohol) was assigned in random order.

Chest electrodes for recording ECG were next attached (CM5 placement) and the sphygmomanometer cuff was put on the left arm. The oncometer was then sealed over the right middle finger. One half hour after the last of the drink had been ingested, the breath sample was taken for determination of the level of blood alcohol. The subject then put on a medium weight sweater to prevent general cooling. Following this he sat in front of the air flow

apparatus and held his forehead on the pad as described above. This position resulted in the area of the face approximating that innervated by the ophthalmic branch of the trigeminal nerve to be exposed to the air flow. Finally the thermocouple was attached to the cheek. The subject was requested to relax and to remain quiet during the test period and if possible avoid falling asleep. Testing began after the subject had been sitting undisturbed for 10 minutes.

The experimental procedure required approximately 35 minutes during which time the several different conditions were imposed. The experiment began with a 3 minute no wind period during which resting measurements of all variables were made. This period was referred to as baseline. Following this, wind at five different temperatures was blown on the subjects faces. The period of each exposure lasted two minutes. Each wind exposure was separated from the next by a control period of varying length without wind during which time the subjects face temperature was allowed to return to the level observed during the initial baseline period. The order of presentation of the different temperatures was randomized. The temperatures used included 33C, 30C, 21C, 15C and 9C. All variables were measured during wind exposure and control periods as previously mentioned.

On completion of the experiment, after the second

breath sample was taken for blood alcohol determination, subjects completed a questionnaire, designed to asses their perceived thermal comfort. Answers were rated on a scale ranging from -3 to +3, negative numbers indicating degree of coldness, positive numbers indicating degree of warmth, zero indicating neutral. This questionnaire and scale were adapted from those developed by Bedford (1958) (Appendix II).

As a safety precaution, subjects were required to remain in the lab until their breath sample measurements declined below 40 mg%.

D. Data Analysis

Statistical analysis of the data consisted of two-way Analysis of Variance with repeated measures for all data except that from the perceptual questionnaire. Analysis on the latter was made using a simple t-test. Statistical significance was accepted at the .05 level in all cases.

IV. RESULTS

A. Blood Alcohol Levels

Blood alcohol concentrations (see Table 11, Appendix I) averaged 85 mg% ± 4 (mean ± sem) when determined at the beginning of the experiment. This had dropped an average of 15 mg% by the end of the experiment to a mean value of 70 mg% ± 3.

B. Face Temperature

The effect of wind of various temperatures on mean face temperature can be seen in fig. 2. Mean face temperature dropped significantly from control values when subjects were exposed to winds of 21C, 15C and 9C in trials where alcohol was not ingested ($p<0.05$). The maximum drop in face temperature was 10.5C ± 1 (mean ± sem) and occurred during the second minute of exposure to a 9C wind. Similar changes in mean face temperature were seen during trials where alcohol had been ingested. Alcohol had no apparent effect on the magnitude or rapidity of this change. Mean face temperature tended to be slightly higher when alcohol had been consumed. This difference approached statistical significance ($p=0.06$).

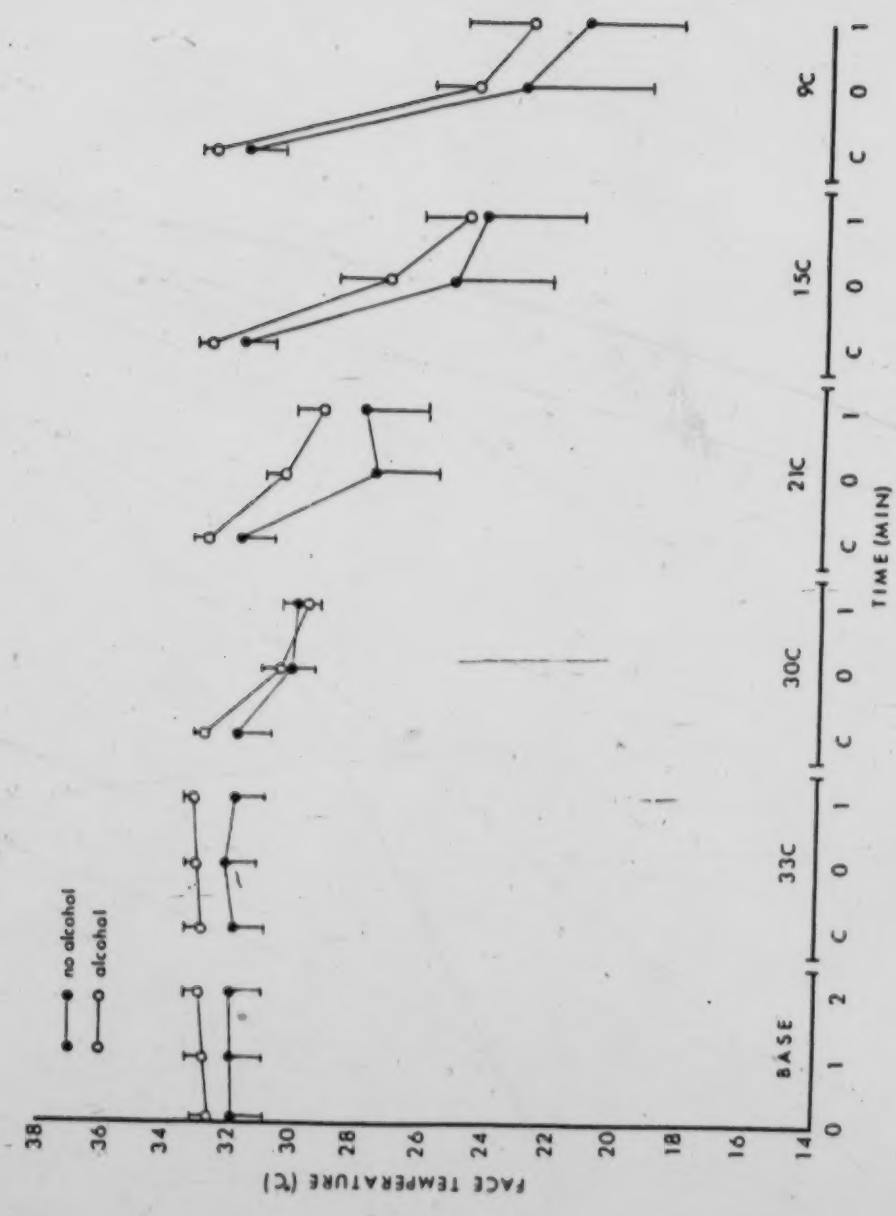

Fig.2. Influence of alcohol ingestion on face temperature in response to 5 wind temperatures. Legend- C=control,no wind, Base=baseline, no wind. All values mean ± sem.

C. Heart Rate

Mean heart rate was not significantly affected when air of any temperature was blown on subjects faces. This is evident in fig. 3.

Since heart rate has been reported to decrease fairly rapidly in response to cold air being blown on the face LeBlanc et al 1975a, Hayward et al 1976) it was decided to examine the beat by beat change in heart rate response during exposure to the coldest wind (9C). This was done to determine if a transient bradycardia had occurred. A transient bradycardia may have been masked by the arithmetical averaging procedure used to calculate mean heart rate. Accordingly, heart rate was determined directly from the chart paper during each 5 second period associated with blood flow measurements and were not combined to give an average for that period. This resulted in a temporal record spanning the last 5 seconds of control prior to wind exposure and the first fifteen seconds of each minute of wind exposure as can be seen in fig. 4. Heart rate tended to decrease from control values after fifteen seconds of wind exposure but this change was not statistically significant ($p>0.05$). Heart rate had returned almost to control values by the end of 75 seconds of wind exposure.

Alcohol had no apparent effect on heart rate although

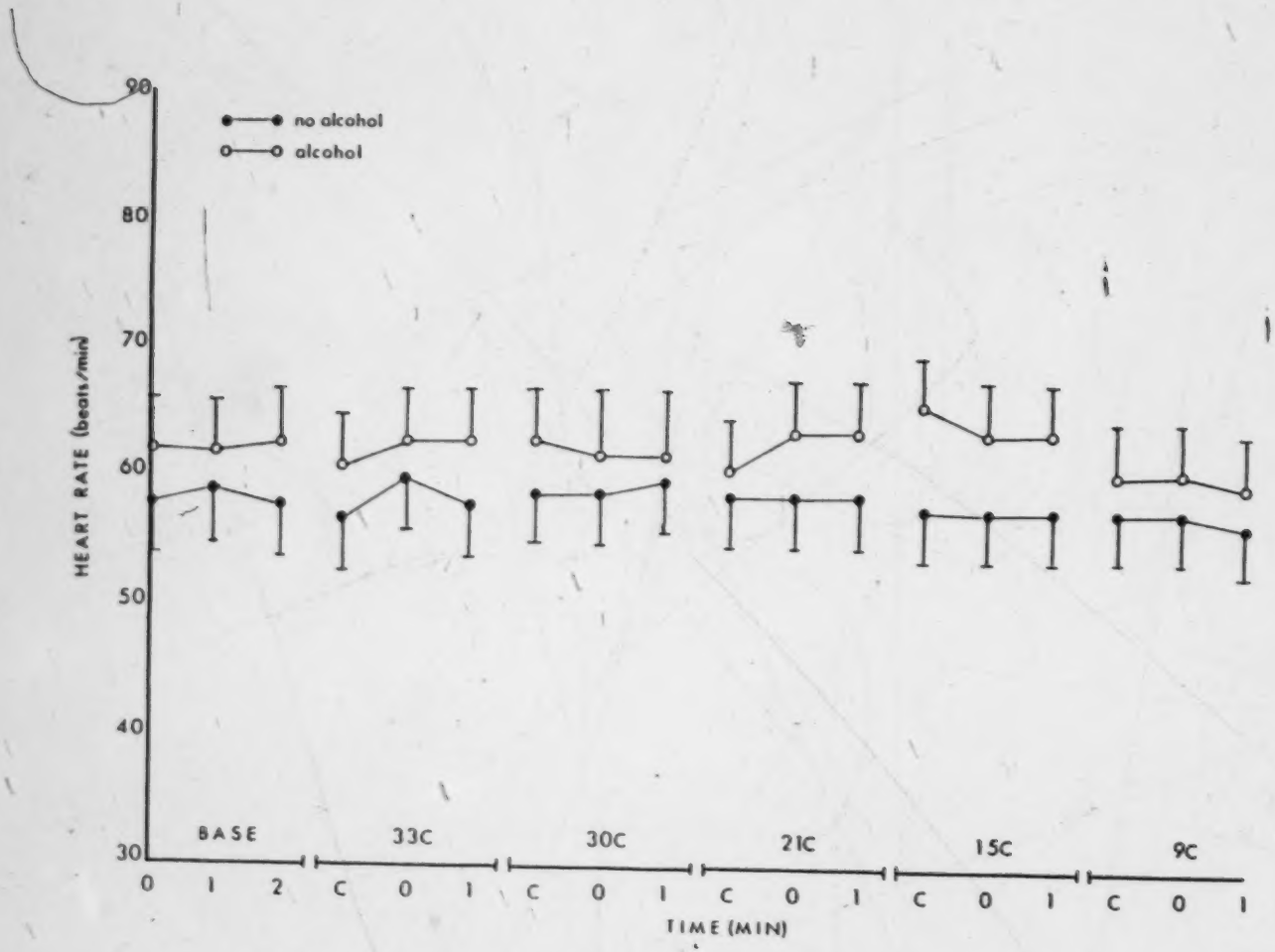

Fig.3. Influence of alcohol ingestion on mean heart rate in response to 5 wind temperatures. Legend- as per fig.2.

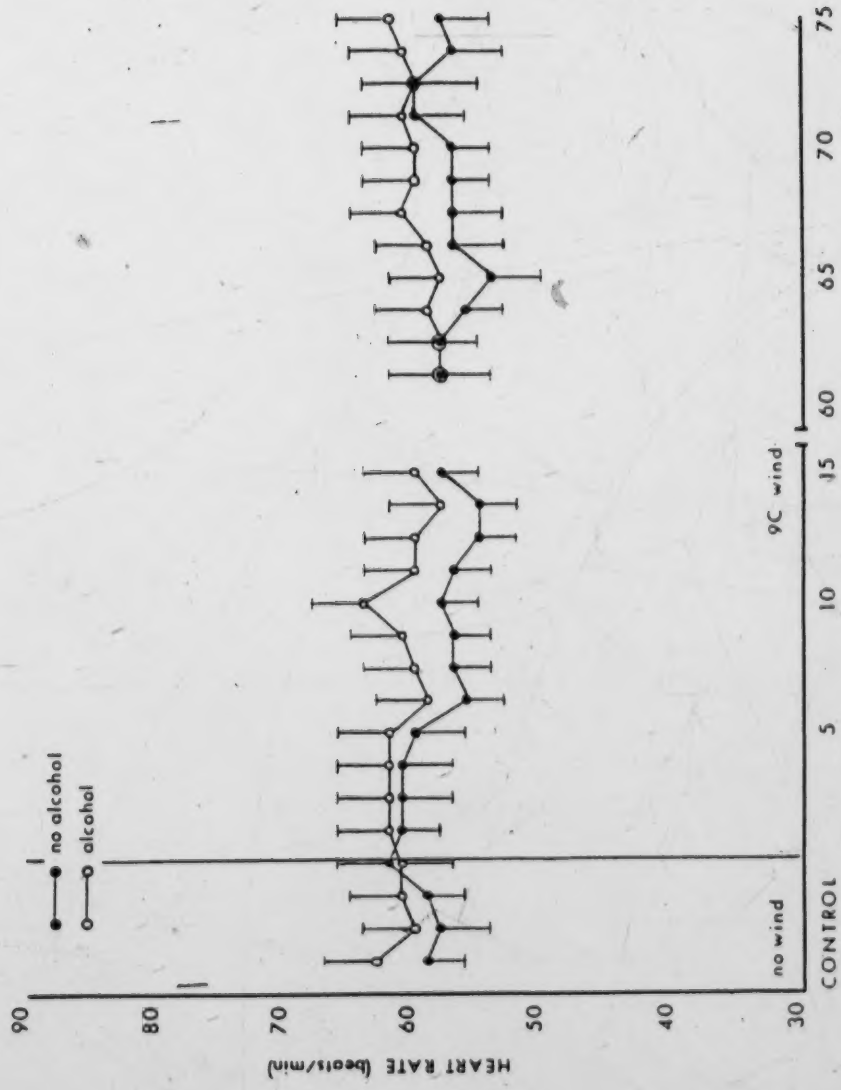

Fig. 4. Influence of alcohol ingestion on heart rate response to 9C wind from the last 5 sec. of control through the first 15 sec of each of two minutes of wind exposure. All values mean ± sem.

the mean values for heart rate tended to be slightly higher. This difference was not statistically significant.

D. Finger Blood Flow

Mean finger blood flow (FBF) tended to decrease immediately from control values when winds of 21C, 15C and 9C were blown on the subjects faces as can be seen in fig. 5. The magnitude of the decrease proved not to be statistically significant ($p>0.05$) however there appeared to be a trend toward a rapid response as is suggested in fig. 5. With the 9C winds, FBF decreased 53% from control values within 15 seconds and 71% from control after 75 seconds in trials when alcohol had not been consumed.

Alcohol had no apparent effect on either the magnitude or time course of the response of FBF. However the magnitude of the response to 15C and 9C air was used, although similar to that observed when alcohol was not ingested, proved to be statistically significant ($p<0.05$). Mean FBF was also significantly higher when alcohol had been ingested.

E. Mean Finger Vascular Resistance

Mean finger vascular resistance (MFVR) did not change significantly in response to exposure to wind at any

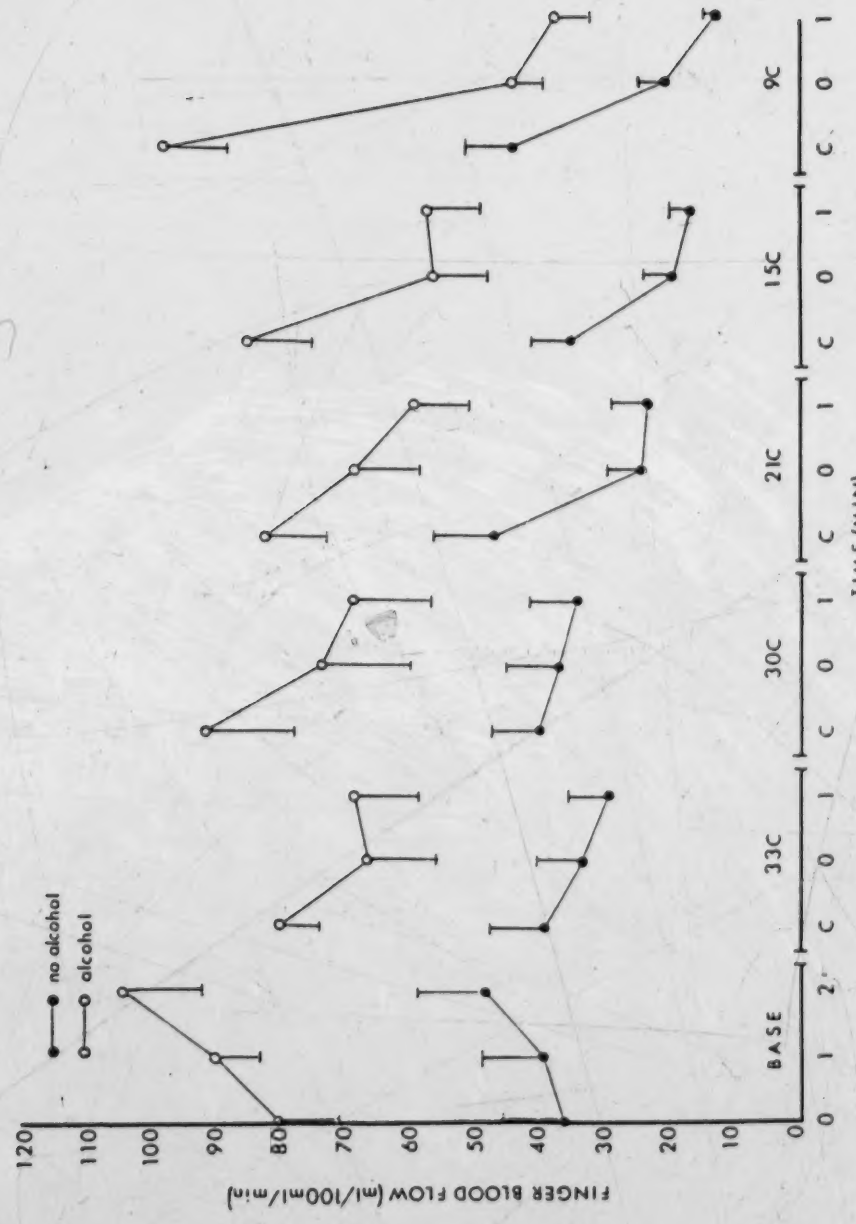

Fig.5. Influence of alcohol ingestion on mean finger blood flow in response to 5 wind temperatures. Legend- as per fig.2.

temperature ($p>0.05$). However it would appear that there was a trend toward an immediate response to the stimuli as can be seen in fig. 6. MFVR more than tripled from control values during the first 15 seconds of exposure to a 9C wind in trials where alcohol had not been ingested. This change approached statistical significance at the 0.05 level ($p=0.06$).

Consumption of alcohol significantly lowered (MFVR) ($p<0.05$). Fig. 6 illustrates that changes in MFVR were much less pronounced in these trials. A similar response time is apparent upon application of a stimulus but the magnitude of the response is so small that it is almost negligible. None of the stimuli evoked a change in MFVR that approached statistical significance in trials where alcohol had been consumed.

F. Arterial Pressure

Mean arterial pressure (MAP) and its components systolic and diastolic pressures were not significantly affected by application of wind on the face as is evident in fig. 7. This was also true when alcohol had been ingested. Average MAP tended to be higher in trials where alcohol had been consumed but this difference was not statistically significant ($p>0.05$).

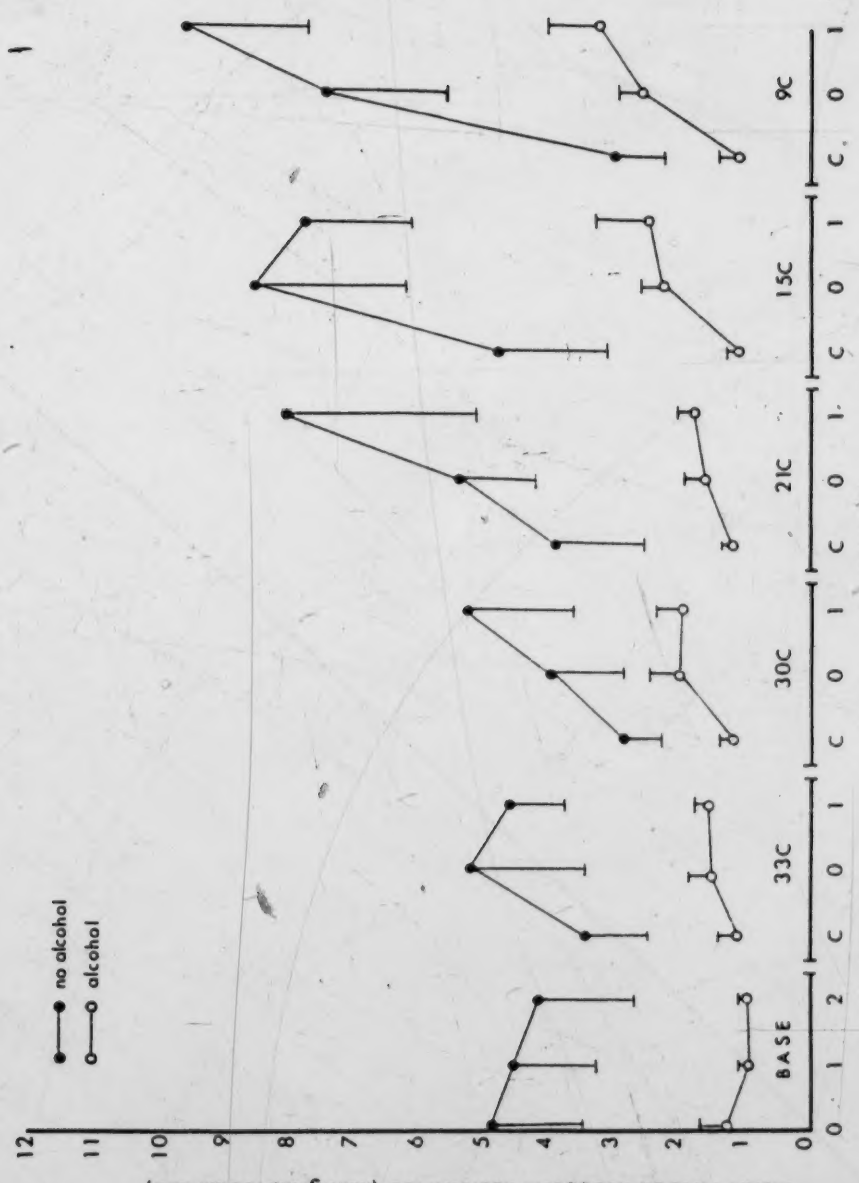

Fig.6. Influence of alcohol ingestion on mean finger vascular resistance in response to 5 wind temperatures. Legend- as per fig.2.

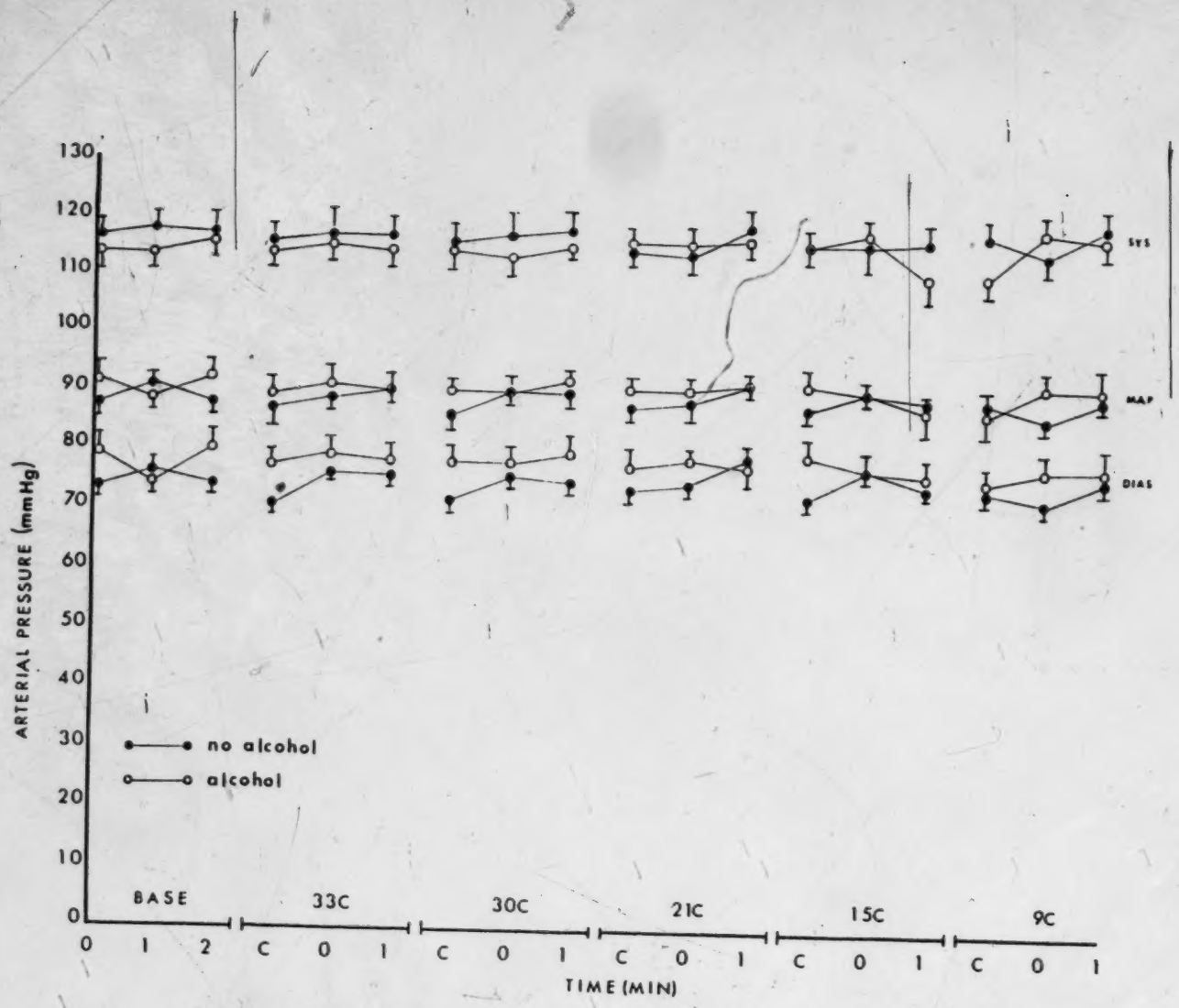

Fig.7. Influence of alcohol ingestion on arterial pressure in response to 5 wind temperatures. Legend- as per fig.2. MAP=mean arterial pressure, SYS=systolic pressure, DIAS=diastolic pressure.

G. Perception of Cold

Analysis of the data from the thermal perception questionnaire revealed subjects reported that they felt warmer when they had ingested alcohol than when they had not. This difference was statistically significant at the 0.05 level.

Although blood flow in the fingers was higher when subjects had ingested alcohol, the subjects did not report a feeling of greater warmth in their hands than when they had not ingested alcohol even though they felt warmer.

V. DISCUSSION

The cardiovascular responses to face immersion have been reported to occur quickly i.e. within 15-30 seconds. For example, Heistad et al (1968) found an average decrease in heart rate of 23% during 30 seconds of face immersion. They also found an abrupt decrease in finger blood flow of 72% within the first 10 seconds of face immersion. Furedy et al (1983) also reported a decrease in heart rate of 5 to 16 beats per minute depending on water temperature during facial immersion. The drop had begun within approximately 7 seconds after immersion and was well developed within 20 seconds.

Similar but less pronounced results have been found when cold air has been used as the stimulus. For example, Hayward et al (1976) reported a significant decrease in heart rate of 6 ± 1% after 30 seconds of wind exposure. Blood flow to the forearm decreased and forearm vascular resistance increased over the same time period. Similar results have been reported by LeBlanc et al (1975a, 1976, 1978).

These results suggest that the cardiovascular responses to application of cold on the face, referred to as the diving reflex, are quite rapid. The actual magnitude of the responses appear to depend on the stimulus modality i.e. water or air.

In view of the above it was somewhat surprising that heart rate did not fall significantly in the present study when air at any of the temperatures used was blown on subjects' faces. Even when the change in heart rate from beat to beat was analysed, no statistically significant changes were found. This lack of response occurred even when face temperature had dropped by 10C.

The tendency for finger blood flow (FBF) and mean finger vascular resistance (MFVR) to drop rapidly (although not statistically significant) is in keeping with the results of Hayward et al (1976) who found a decrease in forearm blood flow of 22% and an increase in forearm vascular resistance of 36% within 30 seconds of exposure to a stream of cold air on the side of the face. The results of the present study indicate that some rapid reflex-like response to the stimulus seems to have taken place. The magnitude of these responses appeared to increase as wind temperature decreased. This was paralleled by a greater decrease in face temperature which suggests that there may be a relationship between degree of face cooling and the magnitude of at least some cardiovascular responses. This would be in keeping with the results of LeBlanc et al (1976) who reported a significant negative correlation (-.76) between cheek temperature and change in heart rate.

The apparent absence of any statistically significant

response in the variables measured even under the coldest conditions in spite of the drop in face temperature in this study may reflect the methods used in presentation of the stimulus. The stimulus was intentionally restricted to a specific portion of the face partly to enhance uniformity and reproducibility and partly because previous studies had implicated the area of the face innervated by the ophthalmic branch of the trigeminal nerve as playing a key role in the elicitation of the diving reflex. In addition, subjects were clothed so as to reduce the chance of their being cooled elsewhere. In contrast, Hayward et al (1976) positioned the outlet of their wind source 6 cm from their subjects faces. Their subjects were only lightly clad (e.g. shorts and no top for males, shorts and bikini top for females). LeBlanc et al (1975b, 1976, 1978) dressed their subjects in snowsuits but allowed the entire face to be exposed to the wind source the outlet of which was positioned 25 cm from subjects' faces. Both authors report significant cardiovascular responses e.g. bradycardia of varying degrees. It may be that exposure of the entire face is required to elicit a significant response when air is being used as a stimulus. This would result in stimulation of a greater number of skin receptors. There may also be a thermoregulatory mechanism operating that takes input from thermoreceptors in other parts of the body. This is suggested from the results of Hayward et al (1976) who reported a decrease in heart rate of 8% when cold was blown onto subjects' abdomens.

The above results suggest that stimulation of the ophthalmic branch of the trigeminal nerve may be insufficient in itself to produce bradycardia when cold air is used as the stimulus. Stimulation of this particular branch of the trigeminal nerve was shown to produce the greatest degree of bradycardia in humans compared to other branches by Khurana et al (1980). These authors reported a 28% reduction in heart rate within one minute when cold was applied to the area of the face innervated by this branch. The stimulus used in this case was a cold pack, temperature 1-2C, placed directly on the face. They also found decreases in heart rate of approximately 11% when areas of the face innervated by the maxillary and mandibular branches of the trigeminal nerve were stimulated individually using the same method. Stimulation of the whole face using cold packs resulted in a drop in heart rate of 21.4% suggesting that the effect of stimulation of all branches simultaneously was not cumulative. This would seem to argue against the need for application of the stimulus to a larger area of the face in the present study however the difference in apparent stimulus strength should not be overlooked. Unfortunately, the above authors did not provide any data on face temperature so it is impossible to assess the degree of face cooling that took place.

Another possible explanation for the lack of any significant decrease in heart rate in the present study

may be the range of resting heart rates observed among the subjects. Five of the ten subjects had resting heart rates below 60 beats per minute. Two of those five had resting heart rates between 40 and 45 beats per minute. These heart rates indicate a resting bradycardia likely caused by fairly substantial vagal activity. It is possible that even the coldest stimulus used in this study was insufficient to stimulate an increase in this already pronounced vagal activity.

Alcohol did not produce any significant effect on most of the cardiovascular responses to the stimuli presented. The lack of effect is not surprising considering the lack of response to the stimuli without alcohol. The only effect observed was an apparent decrease in the magnitude of the response of MFVR. This suggests that alcohol may have attenuated the response of MFVR to the stimuli.

Alcohol influenced subjects' perceptions of thermal comfort. This may have reflected vasodilation in the skin as suggested by higher finger blood flows and somewhat higher face temperatures when alcohol had been ingested. The general perceived increase in thermal comfort when alcohol had been ingested had no apparent effect on the physiological variables measured. This may again be due to the fact that little if any response to the stimuli was seen even without alcohol ingestion.

On the basis of the findings of this study the following conclusions may be drawn.

The stimulus provided under the experimental conditions of dress and room temperature were apparently insufficient to produce statistically significant changes in heart rate, arterial pressure, finger blood flow or mean finger vascular resistance despite a drop in face temperature of 10C when 9C air was blown on subjects faces. These results may be due to the lack of exposure of a sufficient area of the face to the stimulus. In addition, the fact that subjects were warmly dressed and sitting in a fairly warm (21-22C) room may have had an influence on the effectiveness of the stimulus. The methods used in stimulus presentation in the present study differ somewhat from those of others (e.g. Hayward et al 1976, LeBlanc et al 1976) and may account for the lack of significant results.

The responses of finger blood flow and mean finger vascular resistance, while not of statistically significant magnitude, seem to indicate the presence of a fast reflex-like response to the stimuli. The statistical analysis revealed a trend toward significance in the magnitude of change of these variables as wind temperature decreased. This may indicate that the reflex is temperature dependent. This would be in keeping with the findings of other authors who have used varying water

temperatures to elicit the diving reflex. For example, Moore et al (1972) found that face immersion in 5C water produced a much greater bradycardia than did face immersion in 25C or 15C water. Although the stimulus of face immersion is not the same as wind the above results do illustrate the apparent temperature dependance of this reflex in humans.

Alcohol had no significant effect on the cardiovascular responses to air blown on the face. In variables where some response was evident without alcohol (e.g. FBF and MFVR), alcohol appeared not to influence the time course of the responses to the stimulus although there may have been some attenuation of the magnitude of the responses. Alcohol did have a statistically significant effect on subjects' perception of thermal comfort but this did not appear to have any influence on the physiological responses to the stimuli.

The results of this study suggest that the production of a reflex similar to the diving reflex in humans using air as a stimulus may require exposure of a more generalized area of the face to the applied stimulus. It is also possible that this reflex is as much a thermoregulatory reflex as it is related to the diving reflex and may be enhanced by total body exposure to the stimulus. Further studies using more intense stimuli and/or a larger area of stimulus application would be of

interest. It is recommended that untrained individuals be used also.

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APPENDIX I

Data Tables

TABLE 2 MEAN HEART RATE
(beats/min)

Subj	Baseline Min 0	1	2	C	33C Min 0	1	C	30C Min 0	1	C	21C Min 0	1	C	15C Min 0	1	C	9C Min 0	1
ALCOHOL																		
1	54	56	57	60	67	59	57	57	56	57	63	56	58	59	56	57	58	56
2	57	61	69	59	63	61	57	61	59	59	61	61	61	63	62	57	56	58
3	--	42	43	43	42	43	44	41	42	43	47	62	68	53	50	41	44	43
4	41	43	46	40	43	46	47	45	45	43	50	44	46	41	44	42	42	41
5	68	72	72	72	69	72	72	69	71	70	69	72	72	72	68	69	67	69
6	74	78	75	79	79	82	81	86	82	76	86	82	86	86	85	75	79	78
7	64	67	64	64	65	67	66	64	67	64	68	64	68	64	65	65	65	63
8	51	52	51	48	50	52	51	53	45	46	50	48	48	50	49	50	49	52
9	81	80	82	82	84	81	82	78	84	81	77	81	82	82	83	84	81	70
10	71	72	72	66	70	71	72	72	73	67	69	68	68	74	65	73	67	66
MEAN	62	62	63	61	63	63	63	63	62	61	64	64	66	64	64	61	61	60
SEM	4	4	4	4	4	4	4	4	5	4	4	4	4	4	4	4	4	4
NO ALCOHOL																		
1	54	59	55	52	54	53	56	55	63	49	63	58	58	--	--	55	58	50
2	48	50	50	52	51	53	50	51	53	50	47	48	48	49	48	53	50	50
3	44	45	45	45	49	47	47	47	47	45	49	51	49	50	46	48	47	47
4	43	43	48	42	53	47	46	46	47	50	50	42	48	41	41	42	49	46
5	67	63	62	62	63	58	65	62	64	65	58	62	60	60	61	63	57	59
6	67	68	67	64	70	67	69	71	70	67	67	77	62	67	74	67	65	63
7	61	58	61	60	59	59	60	60	60	61	60	61	64	58	55	57	57	58
8	44	46	43	42	44	44	43	44	45	48	44	45	45	44	46	43	47	46
9	70	71	68	70	70	69	68	68	68	72	71	69	68	69	69	73	69	69
10	79	86	85	78	83	81	85	81	80	75	80	77	80	82	85	74	77	83
MEAN	58	59	58	57	60	58	59	59	60	59	59	59	58	58	58	58	58	57
SEM	4	4	4	4	4	4	4	4	4	3	4	4	3	4	5	4	3	4

TABLE 3 HEART RATE - BEAT BY BEAT ANALYSIS
(p 1 of 2) (beats/min)

ALCOHOL

					9C Min 1											
Subj	Last 5 Sec Recovery				5 Sec				10 Sec				15 Sec			
1	42	38	40	42	42	41	46	44	45	42	45	43	47	42	46	42
2	55	54	52	48	56	51	52	48	46	45	48	47	47	49	46	50
3	65	63	67	67	70	68	65	64	69	65	63	66	62	66	--	66
4	79	78	82	76	79	76	75	79	74	78	75	78	77	79	80	83
5	55	53	55	58	55	59	62	64	59	60	61	59	57	54	52	56
6	42	36	38	38	53	49	43	39	39	40	42	--	36	38	38	40
7	68	69	73	73	73	72	65	67	65	69	67	68	65	67	66	66
8	88	83	78	77	76	79	81	83	74	76	78	79	81	84	--	84
9	72	70	65	63	68	69	70	69	65	66	70	75	70	64	59	62
10	60	59	60	61	52	58	57	61	56	54	57	60	56	57	60	54
MEAN	63	60	61	60	62	62	62	62	59	60	61	64	60	60	58	60
SEM	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
NO ALCOHOL																
1	46	44	50	50	55	46	47	47	47	44	50	48	48	44	45	48
2	47	42	41	47	46	49	43	51	46	49	57	48	45	44	43	42
3	61	64	65	61	59	64	62	61	55	54	53	56	56	57	53	58
4	71	70	67	71	69	72	68	61	60	65	61	63	69	58	56	--
5	55	57	56	54	59	60	64	62	57	63	61	62	57	53	51	49
6	44	46	43	--	60	53	47	43	46	48	44	--	51	54	49	47
7	66	67	65	66	59	57	61	56	59	55	56	54	58	53	54	53
8	74	72	73	71	72	73	76	74	69	65	63	66	70	69	68	67
9	72	70	71	76	82	83	81	78	74	72	74	77	70	71	74	80
10	54	52	56	53	52	54	58	51	56	52	47	44	42	48	54	--
MEAN	59	58	59	62	61	61	61	60	56	57	57	58	57	55	55	58
SEM	3	4	3	4	3	4	4	4	3	3	3	3	3	3	3	3

TABLE 3 HEART RATE - BEAT BY BEAT ANALYSIS
(p 2 of 2) (beats/min)

Subj		5 Sec			10 Sec				15 Sec			
ALCOHOL												
1	54	56	53	53	54	56	60	57	58	53	52	53
2	40	39	45	42	44	44	42	45	43	42	41	42
3	40	40	41	38	40	40	38	42	39	40	42	42
4	78	75	83	80	72	75	78	79	76	74	77	76
5	60	65	61	61	61	65	63	60	64	61	58	63
6	51	46	51	49	50	55	49	49	53	55	56	53
7	62	64	63	63	68	69	69	70	72	74	73	75
8	60	56	55	60	53	60	56	55	59	61	64	60
9	70	69	67	69	66	64	62	64	66	66	70	75
10	61	67	66	69	84	81	81	80	79	78	79	80
MEAN	58	58	59	58	59	61	60	60	61	60	61	62
SEM	3	4	4	4	4	4	4	4	4	4	4	4
NO ALCOHOL												
1	47	52	47	43	48	49	54	52	53	51	54	54
2	87	86	82	78	88	84	80	78	94	97	90	75
3	67	67	68	69	71	69	72	69	66	67	67	62
4	62	57	55	55	54	55	57	61	64	62	63	69
5	52	49	49	48	45	44	47	47	60	60	48	44
6	66	64	62	64	59	64	61	61	66	66	64	65
7	55	55	59	55	57	55	53	54	62	65	59	57
8	44	50	45	45	48	44	44	45	46	46	43	46
9	50	52	47	42	48	48	47	49	39	39	37	41
10	49	45	45	45	55	53	50	51	49	49	47	52
MEAN	58	58	56	54	57	57	57	57	60	60	57	58
SEM	4	3	3	4	4	4	3	3	4	5	4	4

TABLE 4 FINGER BLOOD FLOW
(ml/100ml tissue/min)

Subj	Baseline Min 1	Baseline Min 2	Baseline Min 3	C	33C Min 1	33C Min 2	C	30C Min 1	30C Min 2	C	21C Min 1	21C Min 2	C	15C Min 1	15C Min 2	C	9C Min 1	9C Min 2
ALCOHOL																		
1	79	83	104	67	62	62	48	17	18	79	59	38	62	20	9	104	44	18
2	136	122	169	116	90	94	125	137	115	169	87	100	114	82	71	119	49	58
3	75	94	100	100	51	83	86	49	61	87	46	37	69	109	104	71	26	51
4	53	73	100	58	24	36	57	20	38	60	27	38	100	29	31	114	20	31
5	117	113	96	96	152	139	128	122	115	120	150	120	93	87	52	170	85	72
6	49	56	77	58	41	53	67	36	31	54	41	42	57	31	40	77	27	30
7	79	86	67	79	86	96	82	107	61	67	72	78	72	60	78	80	67	51
8	65	97	83	81	78	30	83	105	97	71	87	60	87	49	68	106	68	36
9	49	65	60	63	51	49	44	36	32	59	40	33	60	29	35	27	29	11
10	107	89	201	89	36	51	201	115	130	65	87	59	147	74	98	123	39	28
MEAN	80	89	105	80	67	69	92	74	69	83	69	60	86	57	58	99	45	38
SEM	9	7	13	6	11	10	14	14	12	10	10	9	9	9	9	11	6	5
NO ALCOHOL																		
1	34	26	56	30	18	16	20	21	13	29	13	11	27	--	--	56	29	13
2	77	98	103	65	45	44	45	39	47	103	45	51	73	42	40	67	19	23
3	8	10	8	8	6	11	16	16	6	6	10	3	6	4	8	14	6	13
4	11	6	6	14	6	11	13	7	6	11	8	8	6	6	5	11	6	5
5	72	92	92	92	83	72	80	89	79	73	40	37	58	35	25	83	51	26
6	36	38	47	49	44	43	41	40	45	64	49	35	36	8	19	47	30	14
7	15	15	22	54	58	40	44	31	32	22	16	12	50	36	15	43	32	11
8	35	43	59	22	31	12	59	38	42	58	35	15	26	14	6	52	10	4
9	9	22	31	37	26	21	31	22	25	42	10	32	31	12	19	28	5	7
10	75	54	67	33	23	30	67	79	62	80	28	39	49	26	17	50	28	19
MEAN	37	40	49	40	34	30	41	38	35	48	25	24	36	20	17	45	21	13
SEM	8	9	10	8	7	6	7	8	7	9	5	5	6	4	3	7	4	2

TABLE 5 MEAN FINGER VASCULAR RESISTANCE
mmHg/(ml/100ml tissue/min)

Subj	Baseline Min 0	1	2	C	33C Min 0	1	C	30C Min 0	1	C	21C Min 0	1	C	15C Min 0	1	C	9C Min 0	1
ALCOHOL																		
1	1.1	1	0.8	1.3	1.3	1.3	1.8	4.9	5.1	1.1	1.5	2.3	1.3	4.4	8.8	0.8	1.9	4.6
2	0.7	0.8	0.6	0.8	1	1.0	0.8	0.7	0.8	0.6	1.1	1.0	0.9	1.2	1.3	0.8	1.9	1.6
3	1.2	0.9	0.9	0.9	1.8	1.1	1.1	1.8	1.5	1.1	2.1	2.5	1.6	--	--	1.3	3.6	1.9
4	1.8	1.3	1.0	--	3.6	2.8	1.5	4.5	2.3	1.4	3.3	2.5	1.0	3.2	2.7	0.7	4.6	3.1
5	0.8	0.7	0.9	0.9	0.6	0.6	0.7	0.7	0.8	0.7	0.6	0.7	1.0	1.0	1.5	0.5	1.0	1.2
6	2.3	--	1.4	1.8	0.7	2.0	1.6	3.0	3.4	2.0	2.6	2.6	1.8	3.5	2.7	1.4	4.0	3.7
7	1.1	1.0	1.3	1.1	--	0.9	1.0	0.8	1.5	1.3	1.2	1.1	1.2	1.4	1.2	1.1	1.2	1.6
8	1.3	0.9	1.0	1.0	1.0	2.9	1.0	0.9	0.8	--	0.9	1.4	0.9	1.7	1.2	0.8	1.2	2.4
9	2.1	1.5	1.7	1.6	2.0	2.1	2.4	2.9	3.3	1.6	2.6	3.2	1.7	3.5	3.0	3.8	4.1	10.6
10	0.8	0.9	2.4	1.0	2.4	1.7	0.4	0.8	0.7	1.4	1	1.5	0.6	1.2	0.8	0.7	2.3	2.9
MEAN	1.3	1.0	1.0	1.1	1.6	1.6	1.2	2.1	2.0	1.2	1.7	1.9	1.2	2.3	2.6	1.2	2.6	3.4
SEM	0.2	0.1	0.1	0.2	0.3	0.2	0.2	0.5	0.4	0.1	0.3	0.3	0.1	0.4	0.8	0.3	0.4	0.8
NO ALCOHOL																		
1	--	3.4	1.6	3.0	5.0	5.7	4	4.2	6.8	3.1	6.8	8.5	3	--	--	1.6	3	6.9
2	1.1	0.9	0.8	1.3	2.0	1.9	2.0	2.3	2.0	0.8	2	1.7	1.3	2.1	2.2	1.3	4.8	4
3	11.9	9.1	11.5	11.5	15.2	8.9	5.6	6.3	16.2	15.5	10.1	34.7	15.8	25.3	12.3	7.4	15.7	7.8
4	7.7	15.0	15.3	7	16.8	8.7	6.6	13.9	15.5	9.1	12.3	12.5	15.3	16.5	18.2	8.6	15.2	20.8
5	1.2	1.0	0.9	0.9	1.1	1.3	1.0	0.9	1.1	1.2	2	2.4	1.5	24	3.3	1.0	1.5	3.3
6	2.7	2.5	2.0	1.7	2.1	2.2	2.4	2.4	2.2	1.5	2.0	2.8	2.7	12.4	5.0	2.0	3.2	6.9
7	6.0	6.1	4.2	1.6	1.5	2.2	2.1	2.9	2.8	4.2	5.6	8.1	1.7	2.5	6.0	2.1	2.8	8
8	2.2	1.9	1.3	3.4	2.7	6.8	1.3	2.1	1.9	1.3	2.2	5.9	3.0	6	14.3	1.6	8.1	21.5
9	10.3	4.6	3.1	2.5	3.5	4.4	1.0	4.2	3.7	2.2	9.1	3.0	3.1	7.8	4.8	3.4	19.2	14.3
10	1.2	1.7	1.3	2.5	3.8	--	1.3	1.1	1.4	1.0	3.1	2.2	1.6	3.5	5.4	1.8	2.9	4.3
MEAN	4.9	4.6	4.2	3.5	5.3	4.7	2.9	4.0	5.4	4.0	5.5	8.2	4.9	8.7	7.9	3.1	7.6	9.8
SEM	1.3	1.3	1.5	1.0	1.7	0.9	0.6	1.1	1.7	1.4	1.2	3.0	1.7	2.4	1.7	0.8	2.0	2.0

TABLE 6 MEAN ARTERIAL PRESSURE
(mm Hg)

Subj	Baseline Min 0	1	2	C	33C Min 0	1	C	30C Min 0	1	C	21C Min 0	1	C	15C Min 0	1	C	9C Min 0	1
ALCOHOL																		
1	87	83	85	85	82	79	85	83	92	83	88	87	80	87	79	85	85	83
2	91	91	94	93	90	89	92	91	96	94	94	95	95	95	89	91	92	91
3	90	85	93	93	92	93	91	88	90	93	96	91	107	--	--	91	94	94
4	93	94	101	--	87	99	83	90	89	85	90	93	101	93	84	80	91	97
5	89	92	82	82	89	85	91	79	93	87	88	89	89	88	79	87	86	89
6	112	--	107	104	113	107	105	109	105	109	107	107	105	107	109	107	109	110
7	88	87	89	88	--	83	88	89	91	89	88	88	86	85	90	84	81	81
8	83	83	85	78	83	86	85	89	81	--	82	85	77	84	81	81	81	85
9	101	99	104	99	104	103	105	104	104	97	103	107	104	101	106	102	118	117
10	84	83	87	87	85	85	87	87	90	88	87	89	85	90	82	82	90	82
MEAN	92	89	93	90	92	91	91	91	93	92	92	93	93	92	89	89	93	93
SEM	3	2	3	3	3	3	2	3	2	2	2	2	3	2	4	3	3	4
NO ALCOHOL																		
1	--	87	91	89	90	91	80	89	88	90	88	93	89	93	89	91	87	90
2	87	87	87	87	90	85	92	90	92	87	90	87	91	86	86	86	92	92
3	95	91	92	92	91	98	90	101	97	93	101	104	95	101	98	104	94	101
4	85	90	92	98	101	96	86	97	93	100	98	100	92	99	91	93	91	104
5	84	91	85	85	88	91	83	82	87	84	80	87	84	83	82	79	75	85
6	95	94	95	91	94	95	97	96	99	95	99	98	98	99	94	95	95	97
7	90	92	92	87	88	86	90	90	89	92	90	97	86	91	90	90	89	88
8	77	81	77	74	82	82	77	80	81	76	76	88	78	84	86	82	81	86
9	93	100	95	92	92	93	92	93	93	93	91	96	95	93	91	96	96	100
10	87	94	87	82	88	--	87	88	87	83	86	84	79	91	91	88	80	81
MEAN	88	91	89	88	90	91	87	91	91	89	90	93	89	92	90	90	88	92
SEM	2	2	2	3	2	2	2	2	2	2	3	2	2	2	1	2	2	2

TABLE 7 SYSTOLIC PRESSURE
(mm Hg)

Subj	Baseline Min 0	Min 1	Min 2	C	33C Min 0	Min 1	C	30C Min 0	Min 1	C	21C Min 0	Min 1	C	15C Min 0	Min 1	C	9C Min 0	Min 1
ALCOHOL																		
1	118	119	112	113	109	108	119	112	119	112	119	120	112	125	100	112	119	112
2	112	112	113	112	112	108	113	112	112	113	113	112	110	112	107	110	113	112
3	112	111	120	120	116	119	116	112	119	119	128	121	137	--	--	116	121	121
4	118	121	120	--	120	128	112	120	124	120	119	126	120	121	121	113	120	124
5	106	116	110	110	112	111	112	97	112	112	108	112	112	107	96	107	112	112
6	129	--	128	128	132	128	123	132	124	132	128	128	123	135	128	128	137	129
7	104	100	107	103	--	96	102	107	105	107	105	104	108	112	112	104	108	112
8	104	112	108	103	104	112	108	112	106	--	104	105	105	106	106	106	106	105
9	132	135	132	128	129	132	140	128	128	132	136	137	132	134	135	128	144	144
10	109	107	112	112	112	112	112	116	118	112	116	119	112	119	103	103	120	119
MEAN	114	114	116	114	116	115	115	114	116	117	117	118	117	119	112	112	120	119
SEM	3		3	3	3	3	3	3	2	3	3	3	3	3	4	3	3	3
NO ALCOHOL																		
1	--	120	119	119	120	122	113	120	119	112	119	120	116	--	--	119	120	119
2	112	112	112	111	111	112	118	111	119	112	112	112	112	112	--	111	119	117
3	134	128	129	129	138	135	128	142	136	132	134	144	140	144	137	143	135	144
4	126	129	134	137	142	128	128	138	132	141	137	139	134	129	128	139	132	139
5	105	104	104	104	103	112	105	104	107	103	104	109	109	102	103	107	96	111
6	125	122	118	112	113	112	113	119	120	112	118	119	119	120	119	118	112	116
7	121	116	116	112	113	112	112	112	120	116	119	116	112	112	112	112	112	112
8	103	104	102	104	104	109	102	105	103	103	97	107	105	105	111	103	103	112
9	119	124	123	120	119	122	123	120	119	120	112	126	123	128	119	128	128	132
10	112	121	119	118	116	--	119	112	120	112	113	112	106	118	120	119	112	112
MEAN	117	118	117	116	117	118	116	118	119	116	116	120	117	118	118	119	116	121
SEM	3	3	3	3	4	3	3	4	3	3	3	3	3	4	3	3	3	3

TABLE 8 DIASTOLIC PRESSURE
(mm Hg)

ALCOHOL Subj	Baseline Min 0	1	2	C	33C Min 0	1	C	30C Min 0	1	C	21C Min 0	1	C	15C Min 0	1	C	9C Min 0	1
1	79	72	80	80	80	80	79	76	76	80	84	76	92	--	--	78	80	80
2	80	80	68	68	78	72	80	70	84	75	78	77	77	78	70	77	75	78
3	71	71	74	74	72	71	74	73	76	76	73	74	71	76	72	72	75	64
4	86	81	90	84	92	89	88	92	92	80	87	92	90	84	91	89	105	103
5	72	68	73	65	72	73	73	78	68	--	71	65	63	73	68	68	68	75
6	80	80	80	80	--	76	81	80	84	80	80	80	75	71	79	74	68	65
7	104	--	96	92	103	97	96	97	96	97	97	97	96	93	100	96	96	100
8	80	80	84	84	79	80	81	80	88	84	84	86	87	87	80	81	81	80
9	80	80	91	--	73	84	68	75	72	68	76	76	91	79	73	64	77	80
10	71	65	71	71	68	64	68	68	78	68	72	70	64	68	68	71	68	68
MEAN	80	75	81	78	80	79	79	79	81	79	80	79	81	79	78	77	79	79
SEM	3	2	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	4
NO ALCOHOL																		
1	75	72	74	74	72	80	71	81	77	73	84	84	72	80	79	84	73	80
2	73	80	75	75	80	80	72	71	79	75	68	76	72	73	71	65	65	72
3	75	80	71	64	74	--	71	76	70	68	73	70	65	78	77	72	64	65
4	80	88	81	68	79	76	76	79	80	69	80	81	81	75	77	80	80	84
5	64	70	65	59	71	68	65	68	70	60	65	78	64	73	73	71	71	73
6	75	80	80	75	75	73	79	79	73	80	75	87	73	80	79	76	78	76
7	80	80	84	81	84	86	89	84	88	87	89	87	88	88	81	84	87	88
8	75	75	75	75	79	71	79	79	79	75	79	75	80	73	--	74	79	80
9	65	71	71	78	80	80	65	76	73	80	79	81	71	84	73	72	71	87
10	--	70	77	74	71	75	64	73	72	79	72	80	76	--	--	77	71	75
MEAN	74	77	75	72	77	77	73	77	76	75	76	80	74	78	76	76	74	78
SEM	2	2	2	2	1	2	2	1	2	2	2	2	2	2	1	2	2	2

TABLE 9 FACE TEMPERATURE (°C)

Subj	Baseline Min 0	1	2	C	33C Min 0	1	C	30C Min 0	1	C	21C Min 0	1	C	15C Min 0	1	C	9C Min 0	1
ALCOHOL																		
1	33.1	33.2	33.2	33.2	33.4	33.3	33	30.1	29.8	33.2	31	30	33.1	26.5	24.5	33	--	22.8
2	32.5	32.7	32.9	32.9	33.1	33.4	33.0	30.3	30.1	32.9	31	28.3	33	25.9	--	33.1	24.5	22.3
3	33.1	33.1	33.3	33.2	33.5	33.5	33	31.1	30.1	33.1	30.9	30	32.9	29.3	28	33	26.4	24
4	32.5	32.5	32.5	32.5	33	33.1	32.5	30.2	29.8	32.3	29	28.2	32.5	24.5	22.5	32.5	22.9	19.8
5	33.1	33.3	33.5	33.4	33.5	33.5	33.4	31.3	30.3	33.4	31.3	30.4	33.4	28.7	27.2	33.3	26.7	24.4
6	32.1	32.1	32.3	32.2	33.1	33.1	32.1	30.3	30.3	32.3	30.9	29.7	32.4	27.9	26.2	32.5	26.7	23.5
7	33.3	33.4	33.4	33.4	33.5	33.9	33.3	30.3	29.7	33.4	31.4	30.5	33.3	29.5	28.5	33.2	28.1	26.2
8	33.5	33.7	33.7	33.6	33.3	33.4	33.5	30.5	29.2	33.5	30	29.2	33.5	27.1	25.7	33.7	24.7	21.2
9	31.9	33.4	33.5	33.0	33.5	33.5	33.0	31.5	30.4	33.1	31.4	30.3	33	29.3	28.1	33.2	27.9	25.4
10	32.9	33	33.1	33.1	33.5	33.5	33	31.5	30.4	33.2	31	29.7	33.1	27.4	25.3	33.2	27.5	24.5
MEAN	32.8	33	33.1	33.1	33.3	33.4	33.0	30.7	30	33.1	30.7	29.6	33.1	27.6	26.2	33.1	26	23.4
STD	0.5	0.5	0.4	0.4	0.2	0.2	0.4	0.5	0.3	0.4	0.7	0.8	0.4	1.6	1.4	0.4	1.3	2
NO ALCOHOL																		
1	--	31.9	31.9	31.9	31.7	31.7	32.0	31.4	30.8	31.9	29.7	29.1	31.9	--	--	32	23.5	21.5
2	32.1	32.3	32.5	32.4	33.5	32.9	32.5	30.2	29.7	32.3	30.5	29.4	32.3	28.5	27.5	32.4	25.7	23.4
3	31.2	31.2	31.4	31.3	31.2	31.3	31.2	29.7	29.9	31.2	26.9	26.4	31.2	23.2	22.1	31.3	22.3	20.5
4	31.4	31.4	31.5	31.4	31.7	31.7	31.3	30.3	30.1	31.3	29	28.1	31.3	26.9	25.3	31.4	24.7	22.4
5	31.9	31.9	32	31.9	31.9	31.9	32.0	30.3	30.1	31.8	25	24.7	31.8	18.8	18.0	31.9	18.8	18.0
6	30.9	31	31	31	31.3	31.4	31	30.3	30.1	30.9	27.9	27.3	30.9	24.4	23.2	30.9	23.3	20.7
7	32.7	32.5	32.1	32.5	33.4	32.5	32.4	30.7	30.1	32.3	30.9	30.1	32.3	28.5	27.9	32.4	27.2	25.1
8	32.5	32.7	32.5	32.5	32.2	32.1	32.6	30.1	30.4	32.5	26.5	26.5	32.5	21.9	22.3	32.6	15.5	14.9
9	34.3	34.4	34.7	34.5	34.4	--	34.6	32.2	31.4	34.3	31.7	31.3	34.3	29.3	28.2	34.3	28.5	25.5
10	32.3	32.5	32.5	32.5	33.5	33.5	32.4	30.5	30.2	32.3	30.9	30.1	32.3	29.2	26.9	32.4	27.2	24.1
MEAN	32	32.1	32.2	32.2	32.4	32.1	32.2	30.5	30.2	32.1	27.9	28.3	32.1	25.6	24.6	32.0	23.6	21.6
STD	1	1	1	1	1	1	1	0.7	0.5	1	2	2	1	3	3	1	4	3

TABLE 10 THERMAL COMFORT

Subj	Question A: How Do You Feel? ALC	NO ALC	Question B: How Are Your Hands? ALC	NO ALC
1	-2	-3	0	0
2	-3	-3	0	-1
3	-1	-1	0	-1
4	-2	-2	0	0
5	-1	-1	2	0
6	-3	-3	0	0
7	-1	-3	-2	1
8	-1	-1	1	2
9	1	-1	2	2
10	-2	-2	2	2
MEAN	-1.7	-2.0	0.5	0.5
STD	0.78	0.89	1.15	1.07

TABLE 11 BLOOD ALCOHOL LEVELS (mg %)

Subj	Initial	Final
1	80	60
2	90	60
3	80	70
4	110	80
5	70	80
6	70	60
7	90	60
8	90	80
9	80	70
10	90	80
MEAN	85	70
STD	11.79	9.43

APPENDIX II

Thermal Comfort Questionnaire

A. How do you feel?

-3 Cold
-2 Cool
-1 Slightly Cool
 0 Neutral
 1 Slightly Warm
 2 Warm
 3 Hot

B. How are your hands?

-3 Cold
-2 Cool
-1 Slightly Cool
 0 Neutral
 1 Slightly Warm
 2 Warm
 3 Hot

APPENDIX III

Subject Consent Form

I _____ am willing to act as a subject in two cold air tests to be carried out by Mr. B. Bain in the Department of Physical Education and Sports Studies at the University of Alberta. During the tests I agree to drink a certain amount of ethyl alcohol mixed with orange juice knowing the tests are intended to determine the effect of ethanol on physiological responses to cold air blown on the face. I know I am free to withdraw from the tests any time I wish and agree to withdraw at the request of Mr. Bain should he wish to terminate the test at any time. At the conclusion of the tests I agree to remain in the laboratory until measurement of my blood alcohol indicates 20 mg/100 ml or less.

In agreeing to take part in this study I waive the University of Alberta of any and all legal claims in connection with these tests.

DATE: _____ SUBJECT: _____
(Signature)

SUPERVISING STAFF MEMBER: _____
(Signature)